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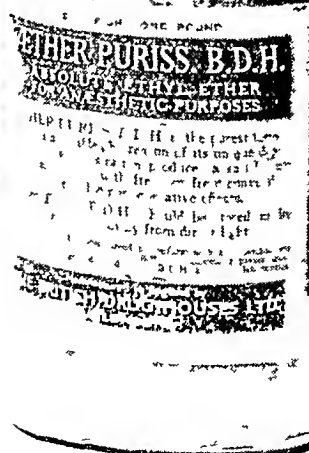
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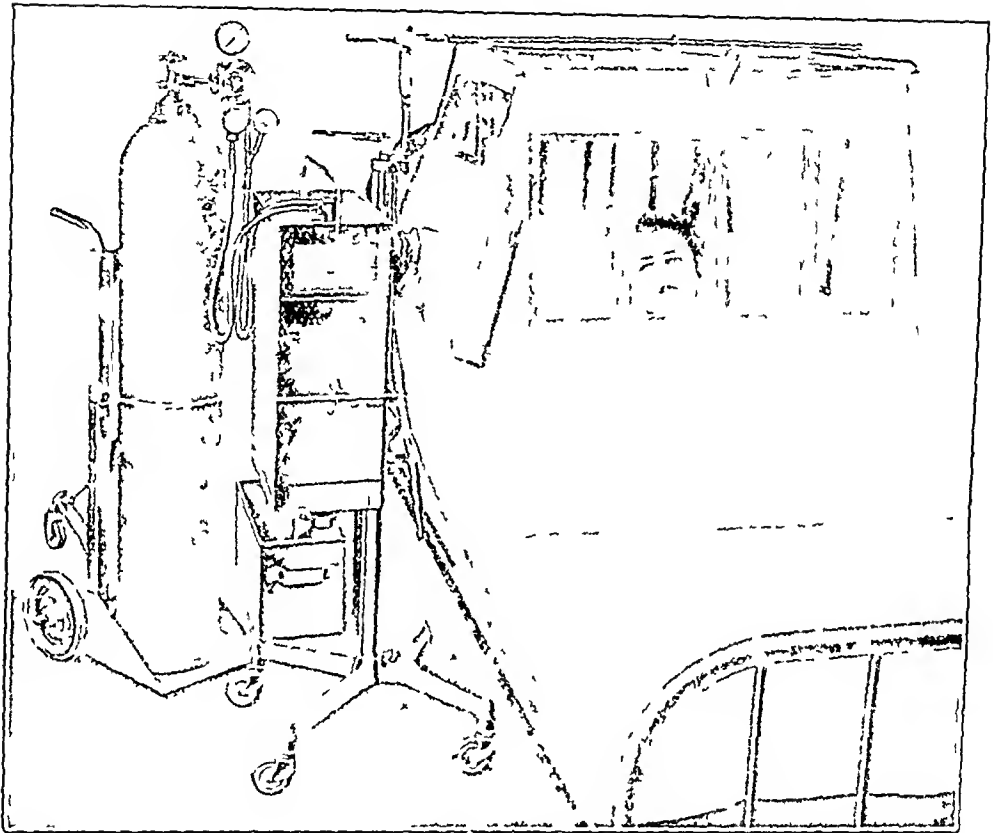
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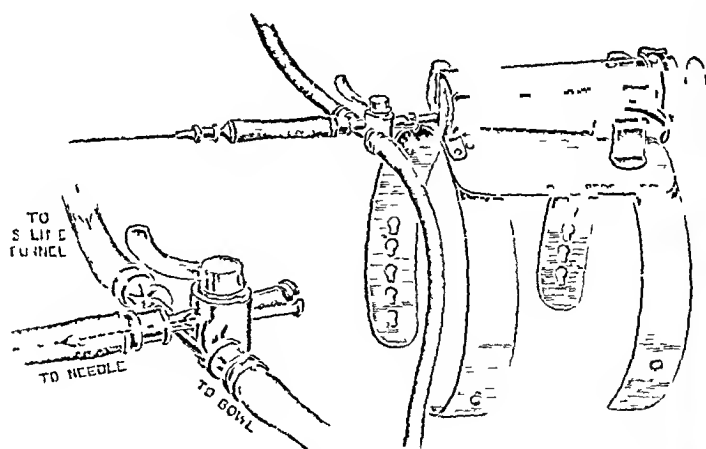
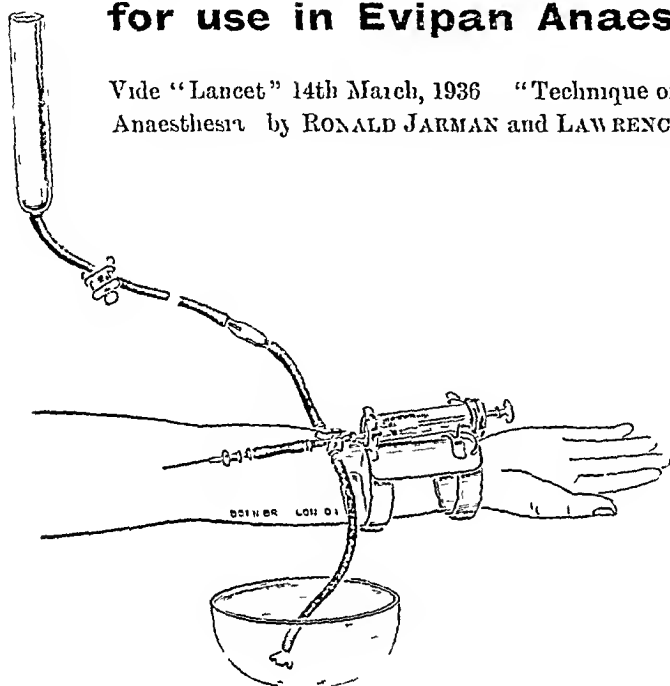
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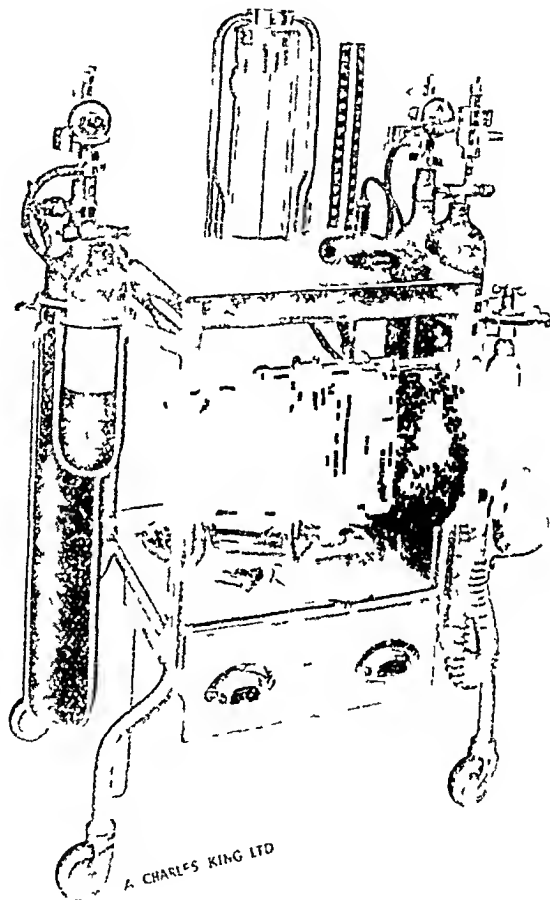
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British Journal of Anæsthesia

VOL. XVI No. 1

OCTOBER, 1938

PENTOTHAL ACID : A NEW BASAL ANÆSTHETIC

By J STEPHEN HORSLEY, M R C S

Senior Assistant Medical Officer at Hernon Hospital

THE introduction of pentothal acid as a basal anæsthetic is the outcome of three years' clinical and laboratory research with the corresponding salt, pentothal sodium, which I have administered personally on more than 2,000 occasions. I observed that pentothal sodium was always effective and satisfactory as an intravenous anæsthetic but that when administered orally its action was less reliable, and it occurred to me that for oral use the free acid might be preferable to the unstable sodium salt. The manufacturers expressed the same opinion, and I am indebted to them for having kindly placed at my disposal the first supply available for experimental use in Great Britain.

In evaluating any new drug the fundamental considerations are, firstly, its chemical composition, secondly, its pharmacological properties, and, thirdly, its worth as shown by controlled clinical trial. These three essentials will be considered in sequence.

Chemical Properties

Pentothal is thio-nembutal and it has the formula ethyl (1-methyl butyl) thio-barbituric acid. The main difference

between the sodium salt and the free acid is one of solubility. Both are lemon yellow powders with a faintly sulphurous odour and a bitter taste.

Pentothal sodium is hygroscopic and must be kept in sealed ampoules, it dissolves readily in water, liberating a gas which smells like acetylene, the solution is unstable and should be used within two hours, furthermore, if any precipitate appears the solution must not be used.

Pentothal acid differs in being relatively stable and only slightly soluble in water, it is, however, freely soluble in absolute alcohol but tends to be precipitated on dilution with water, and it is quite insoluble in aqueous alcoholic solutions below 50 per cent. A concentrated alcoholic solution of pentothal may be diluted to 20 per cent or 10 per cent by the addition of pure glycerine, but the solution is very unstable. The acid is also freely soluble in ether, and this may be emulsified with olive oil for rectal injection. The ethereal solution is also unstable and should be used within three hours of preparation. Pentothal acid is still in the experimental stage, and so far it has been supplied either as the purified powder which must be kept in tightly stoppered bottles or in the form of 4-grain tablets. It is probable that it will be offered shortly for general use in 2-grain capsules.

PHARMACOLOGY

Animal investigations with pentothal acid indicate a wide margin of safety between the minimal hypnotic dose (m h d) and the minimal lethal dose (m l d). Orally, in the rat 30 mgm per kilogram produces narcosis which appears in 30 minutes and lasts from two to three hours followed by gradual recovery with transitory inco-ordination and a staggering gait. There is no stage of excitement during induction or recovery. The m l d by mouth is 200 mgm per kilogram and this causes extreme muscular relaxation followed by respiratory paralysis. Post-mortem examination reveals signs of respiratory failure with no other macroscopic pathological changes in the brain, heart, lungs, liver or kidneys.

In the cat the m h d by mouth is 50 mgm per kilogram, whilst the m l d is 225 mgm. Prolonged anaesthesia for 12

hours in the lactating cat has no appreciable effect on the secretion of milk and no apparent narcotic effect of the kittens. A sub-lethal dose of 135 mgm produces deep narcosis with extreme relaxation of skeletal muscles, loss of sphincter control, loss of corneal reflexes, and marked slowing of respiration. Recovery occurs gradually in 24 hours and during awakening there is transitory ataxia and marked nystagmus.

In man, the action is primarily on the brain-stem and to a less extent on the cerebral cortex. The effect is purely depressant and the euphoria which commonly accompanies pentothal intoxication is attributable to the blocking of painful emotions and the inhibition of higher centres.

Small doses are mildly sedative, medium doses are hypnotic and large doses are anæsthetic. In addition, all these doses are anti-convulsant in varying degree.

The action on other bodily functions is slight and probably non-specific. There is no significant change in the pulse or blood-pressure. Respiration is diminished in volume by full anæsthetic doses but is practically unaffected by the dosage recommended for basal anæsthesia.

The action varies with the method of administration only in so far as this increases or delays the rate of absorption. This variation in onset is of such practical importance that it merits special consideration. When the free acid is given orally the period of action is divisible into four fairly well-defined stages.

(1) The pre-absorption stage in which there is no discernible effect averages one hour but in a few instances it shows a wide variation, the limits being 10 minutes to three hours.

(2) The stage of drowsiness is brief, five to ten minutes, and is characterised by slight ataxia, a minor degree of incoherence and confusion, but mainly by an overwhelming desire to sleep.

(3) Basal narcosis. This stage lasts from one to two hours and is suitable for the induction of general anæsthesia. It is characterised by deep sleep, deep regular breathing, absent corneal reflexes, partial analgesia and some degree of surgical relaxation. This stage occurs only when full doses

of pentothal are given and with smaller doses there is merely a prolongation of drowsiness from which the patient can be roused as from natural sleep

(4) The post-anæsthetic stage is one of drowsiness lasting for a few hours, but with less tendency to restlessness than most basal narcotics

The delay in onset occurs only when the drug is given in tablet or powder form, and it seems to be due to the local action of the free acid on the pyloric reflex. It can be reduced by prescribing the drug in capsule form or in a strongly alkaline mixture

Anti-convulsant Action

In addition to observations to be described in 20 epileptics further studies were made to determine the possible antagonism between pentothal acid and cardiazol which is being used therapeutically for inducing convulsions in cases of schizophrenia

The anti-convulsant action was demonstrated and standardised by first determining the dose of cardiazol which when injected intravenously would induce a major epileptiform convulsion. As the dose of cardiazol varies slightly from day to day a convulsion was induced in each patient on three successive occasions and the maximum dose was used subsequently for the test. It was found that a full basal anæsthetic dose of pentothal inhibited twice the therapeutic convulsant dose of cardiazol

Toxicity

The toxicity of pentothal when given orally or rectally is about half that of nembutal and the dose is proportionately larger, and yet the rate of excretion is more rapid than that of nembutal. This difference is due to the sulphur molecule which appears to facilitate the destruction of pentothal in the body

Repeated daily doses were given for a month to 20 epileptics. In each of these cases the usual medication (either bromides or pheno-barbitone) was stopped gradually and replaced by pentothal acid in doses of gr. 11 three times a day. There was a slight reduction in the number of fits in 17 cases, but in three the number of fits was increased. All of them

were alert and able to continue their usual occupations. This group was studied particularly to discover possible secondary pathological effects of pentothal, it is, therefore, satisfactory to record that repeated observations failed to reveal any evidence of local irritant action, of disturbance of renal function, or of change in the leucocyte picture.

In a further small series of 10 patients I studied the effect of acute pentothal intoxication induced by repeating full hypnotic doses at four-hourly intervals for periods varying from three to ten days. Each of these patients (previously restless and excited) slept almost continuously for the number of days stated, and in consequence required special nursing attention for feeding and personal hygiene. There is only a very slight tendency to cumulative action and no evidence of acquired tolerance, nor do withdrawal phenomena appear on abruptly discontinuing the drug. A minor feature observed in three cases was excessive sweating during narcosis and one of these patients had subsequent peeling of her hands and feet. There was no ketosis or evidence of disturbed carbohydrate metabolism, moreover, in every case the therapeutic effect of the prolonged sleep was beneficial.

CLINICAL USE OF PENTOTHAL ACID

The aim of basal anæsthesia is to induce safely and pleasantly a state of drowsy restfulness as a preliminary to complete surgical anæsthesia. The drugs used for pre-anæsthetic medication are of three main types: the compounds of morphia, atropine, and hyoscine, the paraldehyde and avertin group, and, perhaps most popular of all, the barbiturates. The suitability of a barbiturate as a basal narcotic depends mostly upon its reliability of action and its rate of detoxication or elimination. This is of practical importance in preparing a list of patients for operation. Even with the best technique there is always the possibility of unavoidable delay which with very short-acting hypnotics may mean that the premedication effect has worn off to the detriment of smooth anæsthesia. On the other hand, the longer acting barbiturates are more likely to give rise to post-operative restlessness. Both risks are reduced markedly with

pentothal acid which, although given in relatively larger doses, is eliminated more rapidly than other barbiturates

Pentothal acid has been given orally as a basal anæsthetic in an experimental series of 500 cases. In the first 100 it was the sole pre-anæsthetic agent used and the dose was calculated as one grain per stone of bodyweight. It was given one hour before the induction of general anæsthesia. The result of this dose was to induce sleep in 30 per cent and drowsiness with partial amnesia in a further 60 per cent. The remaining 10 per cent were still fully awake. The 10 who failed to respond to this dose were all of the anxious type with signs of autonomic imbalance and a tendency to sympathetic over-activity. This type, as well as patients with hyperthyroidism, have been found to require much larger doses to induce a reasonable depth of narcosis.

The next step was to determine the effect of considerably larger doses. Accordingly, a further series of 200 patients received doses of two grains per stone bodyweight, exceptions being made in cases of obesity, debility and old age where it was considered safer to limit the dose to a maximum of 12 grains. In most of this series the pentothal was given two hours before induction of anæsthesia and for comparative purposes 100 received pentothal acid together with 1/100 gr atropine whilst the other 100 received pentothal alone. The response with or without atropine was so similar that these cases will be described together. The higher dosage gave decidedly better results and yet one perplexing feature recurred with disconcerting frequency. This was the marked variation in time of onset which when investigated showed that two distinct types of response may occur. Thus, even with high dosage it was found that although 35 per cent of patients were sound asleep within half an hour, the remaining 65 per cent were still awake at the end of two hours and in this latter group the maximum effect did not appear until three hours after administration. A similar but perhaps less striking delay in onset is well known with other barbituric derivatives, and it has been attributed to apprehension with resultant gastric retention and consequently delayed absorption in the small intestine. For this reason many anæsthetists favour combined pre-medication with a

barbiturate by mouth and a hypodermic injection of morphia or omnopon and scopolamine

The combined pre-anæsthetic medication of pentothal acid gr xii and morphia gr $\frac{1}{4}$ th was given to the fourth group of 100 patients. Both drugs were given two hours before induction of anæsthesia and the results were good. Ninety-eight per cent were either asleep or else very drowsy and amnesic—the remaining two, both incidentally feeble-minded persons, became excited. Every patient in this series was breathing evenly with no sign of cardio-respiratory embarrassment and in each instance the induction of complete anæsthesia with intravenous pentothal sodium was smooth and satisfactory whilst the recovery was invariably quiet and uneventful.

In the fifth series of 100 patients pentothal acid was again the sole pre-anæsthetic agent but this time it was given in divided doses and the results were notably better than those already described. The scheme of dosage was to begin medication with pentothal gr. viii three hours before the induction of complete anæsthesia and to repeat this dose once or twice at intervals of one hour. This scheme is physiologically preferable to one which bases the dose according to a single factor such as bodyweight, and it makes adequate allowance for individual variations in the depth of narcosis. I observed that two doses of gr viii given three and two hours before induction gave consistently better results than a single dose of gr xxi, and 74 per cent required no further premedication. The remaining 26 per cent were still awake two hours after the commencement of medication and, therefore, according to scheme, received a third dose of gr viii. By the end of three hours every patient was in a deep sleep, with a good pulse and regular breathing, the colour was good in every case, in fact most patients were definitely flushed.

Anæsthesia was completed in this series in 50 cases with pentothal sodium and in 50 with vinyl ether. The induction was invariably smooth, satisfactory and uneventful, but it necessitated special caution on account of the exceptionally small quantity of additional anæsthetic which was sufficient to give deep surgical anæsthesia. In this series there was

no excitement or restlessness during any stage and the only complication was post-anæsthetic vomiting in three cases

Comparison with other Barbiturates

It is instructive to compare pentothal acid with other well-known barbiturates. For this purpose similar groups of 100 patients were given oral pre-medication with soneryl sodium, nembutal, and pentothal sodium respectively. Soneryl sodium was found to be a safe basal narcotic which when given by mouth could be relied upon to produce sleep or drowsiness in 90 per cent of cases, but in the remaining 10 per cent its action was uncertain and appeared to cause increased restlessness on waking. Nembutal was given orally in doses of gr 111 one and a half hours before induction. Its action resembled that of soneryl in being effective in about 90 per cent of cases. When supplemented by morphia gr $\frac{1}{4}$ its action was effective in 99 per cent but even then there was a tendency to post-narcotic restlessness in some 10 per cent. The main difference between pentothal acid and these "heavy" barbiturates was its speed of elimination and the absence of any excitement during recovery.

A final comparison was made between pentothal acid and the sodium salt. Pentothal sodium by mouth is the quickest acting and most powerful barbiturate yet available. In an experimental series of 100 cases each received an initial dose of 0.5 gramme in a small tumbler of water. This produced sleep in 10 to 15 minutes in 80 per cent, and within an hour in 98 per cent.

Those patients who were asleep within 15 minutes were all at the optimal depth of narcosis to ensure a smooth induction of complete anaesthesia. In those cases (about 20 per cent) who are still awake the same dose may be repeated after 20 minutes. Even this is sometimes ineffective, the probable explanation being that the salt is broken down by an acid gastric juice. This dosage is very cautious and safe, and I have given many patients 2 grammes orally without any ill-effect. Moreover, this dose is often sufficient, without any additional anaesthetic, for the performance of examinations, manipulations and minor operations. The only serious

disadvantage of pentothal sodium by mouth is that in 10 per cent of cases its action is too unreliable for general use

Pentothal acid compares favourably with the basal narcotics in general use. It is markedly hypnotic but less sedative than nembutal or soneryl, paradoxically it produces less post-narcotic restlessness. The main difference is its quicker elimination and the calm recovery which resembles closely the awakening from natural sleep

SUMMARY

(1) Pentothal acid is a satisfactory "basal hypnotic" remarkable for its rapid elimination and calm post-anæsthetic awakening

(2) Pharmacological investigations in animals show a wide margin of safety, the therapeutic quotient in cats being 4.5 and in rats 6.6

(3) Pentothal acid is strongly anti-convulsant and to some extent anti-epileptic

(4) The action of pentothal acid by the mouth is made more certain by administering the drug in capsule form together with an alkaline mixture

(5) The most satisfactory scheme of dosage is to give 8 grains (4 capsules) three hours before operation, and to repeat the dose if necessary, after an interval of one hour. No other premedication is required but atropine gr 1/100 may be added if indicated

(6) Pentothal acid is not regarded as a complete anæsthetic. It is used as a basal narcotic to be supplemented as required by intravenous anæsthesia with pentothal sodium or evipan, or by inhalation anæsthesia with nitrous oxide and oxygen, or with vinyl-ether

ACKNOWLEDGMENT

I am indebted to Dr P. W. Bedford, Medical Superintendent of Herrison Hospital, for allowing me facilities to conduct this research. Special thanks are due also to Mr O. T. Moore for having performed all the routine laboratory tests in the above investigation

THE STUDENT ANÆSTHETIST

By PHILIP AYRE, M R C S (Eng)

Anæsthetist to Royal Victoria Infirmary, Newcastle-upon-Tyne, Anæsthetist to Thoracic and Neurological Departments at Newcastle General Hospital, Hon Anæsthetist to Babies' Hospital, West Parade

ON more than one recent occasion the somewhat startling suggestion has been made to the writer that "open" ether should be administered as a routine in hospital, in order that medical students may obtain more practice in the practical administration of this form of anæsthesia. Adequate comment seems difficult and perhaps superfluous, but it is evident that the whole problem of clinical teaching in anæsthetics requires a thorough overhauling in the light of present-day conditions.

The revolutionary changes that have taken place in anæsthesia during the last twenty years have made the task of the clinical teacher of anæsthetics very difficult. The honorary surgeon rightly demands from the specialist anæsthetist all the skilled resources of modern anæsthetic administration, so that patients in hospital may be operated upon with the maximum of efficiency and expedition. As a result, it is becoming an increasingly frequent complaint among medical students and newly qualified practitioners that too much of their anæsthetic course is devoted to using advanced methods and complicated apparatus, so that when they leave hospital they have had little or no experience in the ordinary "open" methods of administering chloroform and ether.

Whether the medical practitioner of to-day is morally justified in anæsthetising his patients with "open" chloroform-ether, when better and more pleasant methods are available, is distinctly open to question. The customary

somewhat unconvincing explanation given is that the general practitioner has no time to use elaborate gas-oxygen apparatus, or, alternatively, that any anæsthetic other than "open" ether is merely a passing craze and will rapidly go out of fashion. This latter theory is especially popular with a few of the older surgeons, who are wont to make sentimental and slightly amnesic after-dinner speeches about the "good old days", when the success of the anæsthetic depended entirely upon the personal skill of the administrator, and nobody ever vomited or had post-anæsthetic pneumonia! It is interesting to observe that the same surgeons may be found next day operating with every appearance of satisfaction, while the anæsthetist administers endotracheal cyclopropane to a patient whose abdomen can scarcely be seen to move, so efficient is the carbon dioxide absorption technique of modern times. As for vomiting or other unpleasant post-operative complications well, ask the patient.

However, to return to the subject under discussion, it would be idle to deny that there are many occasions when for various reasons "open" ether may be the only form of anæsthesia available, and it is certainly essential that every doctor should be able to use this method with reasonable skill and safety to the patient. How, then, is it possible for the clinical teacher of anæsthetics to satisfy the demands of the surgeon and medical student, not forgetting the unfortunate patient who is liable to become a chopping block for both?

It is the practice of the writer, and no doubt every other hospital anæsthetist, to insist upon three essential principles when teaching students

- (1) The patient's airway must at all times be kept free and unobstructed
- (2) Sufficient air or oxygen must be given to prevent anoxia
- (3) The student must know exactly in what stage or plane of anæsthesia the patient is at any given moment

The first two principles are sufficiently obvious to need no further explanation, although it is surprising how frequently

they are neglected in practice. In the writer's experience, the major difficulty which confronts the beginner is to gauge with any degree of accuracy the exact depth of anæsthesia in an anæsthetised patient. To make matters more perplexing, there is often a great lack of uniformity in the clinical instruction on this point, so that the student wanders from one clinic to another, becoming more and more confused by the diversity of the teaching and the multiplicity of the methods demonstrated to him. Indeed, in a busy general hospital there may be as many "systems" of teaching as there are anæsthetists, which is very bad from the student's point of view.

The recent work of Arthur E. Guedel has thrown a welcome light on this problem, and his monograph on the principles of inhalation anæsthesia should be read and re-read by everyone engaged in the administration of anæsthetics. Though occasionally a trifle dogmatic, Guedel's teaching provides a sure foundation upon which the novice may build up a sound and reasoned knowledge of the clinical signs and symptoms of general anæsthesia. For teaching purposes, the present writer employs a composite and slightly modified version of the original Guedel charts. This composite chart is equally applicable to "open" or "closed" methods of anæsthesia. It does not pretend to include everything about anæsthesia, but at least it rescues the student from the bewildering uncertainty which besets him during his early efforts, when every patient either vomits on the operating table or else ceases to breathe.

The clinical teaching of anæsthesia on the lines suggested by Guedel solves many of the difficulties referred to earlier on. Even if the student does spend a large part of his time watching the flowmeters of the gas-oxygen apparatus, he may still be taught to look for and recognise the various signs and planes of general anæsthesia. Moreover, he soon learns that there are certain fundamental principles common to all forms of inhalational anæsthesia. Once he has grasped these principles, he can apply them to whatever anæsthetic he may be called upon to administer, whether it should be endotracheal nitrous oxide-oxygen-minimal-ether or the "open" chloroform-ether sequence. Even cyclopropane

CLINICAL SIGNS OF ANÆSTHESIA (modified from GUEDEL)

FIRST STAGE (Disordered consciousness)		SECOND STAGE (Delirium)	THIRD STAGE OR SURGICAL ANÆSTHESIA		FOURTH STAGE OR OVERDOSE
			FIRST PLANE	SECOND PLANE	THIRD PLANE
RESPIRATION	<p>Patient may hold breath due to reflex irritation from anæsthetic, or nervousness</p> <p><i>Note</i>—This is the most common cause of death during the early stages of anæsthesia, as the patient may suddenly take a deep breath and inhale a lethal concentration of anæsthetic, <i>especially if chloroform is being administered</i></p>	<p>Patient may hold breath due to reflex irritation from anæsthetic, or nervousness</p> <p><i>Note</i>—This is the most common cause of death during the early stages of anæsthesia, as the patient may suddenly take a deep breath and inhale a lethal concentration of anæsthetic, <i>especially if chloroform is being administered</i></p>	<p>REGULAR "AUTOMATIC" RHYTHMICAL RESPIRATIONS</p> <p>Volume of respiration is increased in first plane, becoming shallower in second and third planes</p> <p>Expansion of thorax synchronises with descent of diaphragm, may be felt by placing hand on patient's chest</p> <p>Present in first and second planes, unless patient has emphysematous chest wall</p>	<p>REGULAR "AUTOMATIC" RHYTHMICAL RESPIRATIONS</p> <p>Volume of respiration is increased in first plane, becoming shallower in second and third planes</p> <p>Expansion of thorax synchronises with descent of diaphragm, may be felt by placing hand on patient's chest</p> <p>Present in first and second planes, unless patient has emphysematous chest wall</p>	<p>SHALLOW "SNATCHING" RESPIRATIONS</p> <p>Complete intercostal paralysis No movement of thorax</p> <p>Breathing entirely diaphragmatic, and "gaspings" in character</p>
COLOUR	<p>Should be normal, but patient may become cyanosed due to breathing holding</p>	<p>Should be normal, but patient may become cyanosed due to breathing holding</p>	<p>Should be normal in all three planes</p> <p>Patient may be flushed with ether anæsthesia, due to vasodilatation</p>	<p>Should be normal in all three planes</p> <p>Patient may be flushed with ether anæsthesia, due to vasodilatation</p>	<p>Pallor and "clammy" perspiration</p>
PUPIL REACTIONS	<p>May be dilated due to fear or struggling</p>	<p>May be dilated due to fear or struggling</p>	<p>Pupils are small with divergent squint</p> <p>Pupils still small No squint</p>	<p>Pupils are small with divergent squint</p> <p>Pupils still small No squint</p>	<p>Widely dilated pupils with no reaction to light</p> <p>May be turned upwards under upper eyelid</p>
REFLEXES	<p>Superficial reflexes present, i.e. skin, swallowing, cough and eyelid reflexes</p>	<p>Superficial reflexes present, i.e. skin, swallowing, cough and eyelid reflexes</p>	<p>Superficial reflexes disappear</p> <p>General muscular relaxation</p>	<p>Superficial reflexes disappear</p> <p>General muscular relaxation</p>	<p>Sphincters paralysed</p> <p>Great circulatory depression with fall in blood-pressure</p>

CLINICAL SIGNS OF ANÆSTHESIA

The signs and stages of general anæsthesia, as charted above, are valid for all inhalational anæsthetics. They also apply to all methods of administration, whether by "open mask" or gas and oxygen apparatus.

Preliminary administration of intravenous drugs such as Evipan or Pentothal Sodium, or Avertin per rectum, carries the patient nearly to the end of the SECOND STAGE. Subsequent anæsthesia proceeds according to schedule, but a partial rebreathing technique with nitrous oxide-oxygen-ether should be employed, in order to counteract the respiratory depression resulting from the use of a basal narcotic.

Premedication with Morphine may have a varying effect on the volume of respiration and the amount of pupillary dilatation, but will in no way alter the course of anæsthesia or the sequence of the various stages and planes. If Atropine alone has been administered, the pupils may be widely dilated throughout all stages, this is commonly seen in children.

Whatever anæsthetic technique may be employed, the patient's airway must at all times be kept free and unobstructed. The jaw must be held forward and an artificial airway inserted when necessary. The patient must always be given sufficient air or oxygen to maintain a good colour.

NEVER continue the administration of the anæsthetic if the patient is not breathing, even when you think the patient is only "holding his breath". Remove the mask from the face and allow one breath of air, before continuing the anæsthetic.

In emergency ACT PROMPTLY

EMERGENCY RESUSCITATION

1—Lower the head of the patient, and commence artificial respiration immediately.

2—Pull forward the patient's tongue and make sure there is no mechanical obstruction to respiration (*e.g.* false teeth, or a gauze pack in the pharynx).

3—Inflate the lungs with oxygen or a mixture of oxygen and 5% carbon dioxide. The best way to do this is to pass an endotracheal tube into the trachea.

4—Continue artificial respiration and feel the pulse. If the pulse is still going, inject 5 c.c. of Lobeline or Coramine intravenously, this stimulates respiration.

5—If the pulse has stopped, inject 5 minims of 1/1000 adrenalin into the muscular wall of the right auricle. Use a long needle (5 inches) and make the injection in the third right intercostal space, directing the needle downwards and towards the mid-line. In the adult the heart lies $3\frac{1}{2}$ to $4\frac{1}{2}$ inches below the surface, in a child the depth is about 2 inches.

6—If the heart does not commence to beat within four minutes, ask the surgeon to make an incision through the diaphragm and apply direct cardiac massage. In children this can be done without opening the diaphragm by doubling up the child, and squeezing the heart sub-diaphragmatically through the abdominal wall.

follows the same path as the others, although admittedly the extreme potency of this agent may produce changes with such insidious rapidity, that the clinical signs are sometimes difficult to detect

Naturally, the student must be given a reasonable amount of practice in the administration of "open" ether, but this practice may be strictly limited to suitable cases and not inflicted upon all and sundry, as is sometimes suggested by hospital teaching authorities who ought to know better. After all, the first duty of the anæsthetist is not to the surgeon or the student, but to the long-suffering and often abused patient—a point which occasionally seems to be overlooked in these days of super-efficient organisation and mass production hospitals

To sum the matter up, the time has arrived when an attempt should be made to standardise the clinical teaching of anæsthesia, and a great step forward would be taken if some responsible body—such as the Association of Anæsthetics—were to take the matter in hand, and draw up a scheme which could be used by every clinical teacher in the country. After all, the clinical signs and planes of general anæsthesia are essentially the same for all anæsthetic agents, so why not adopt a uniform method of teaching them? Finally, although every student should be able to administer a reasonably skilful "open" chloroform-ether anæsthetic in emergency, he should most certainly be allowed to use the newer anæsthetics under proper supervision, so that he may learn to appreciate the advances made in anæsthesia, and the difference they make to the comfort and subsequent welfare of the patient

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ANÆSTHESIA AND THE LAW.

By H GRANTHAM DODD, M B , B S , D.A.

IN the *British Journal of Anæsthesia* (July 1938, p 175) Mr F W Fullerton asks for information as to the legal position with regard to the responsibility of the surgeon and the anæsthetist during the performance of an operation under an anæsthetic, so far as the anæsthetist is concerned. In so doing he has raised a matter, or rather a host of matters, of much interest and importance.

The first point to understand clearly is that there is no established legal position, or (to employ the term which appears later in Mr Fullerton's letter), there are no definite legal rulings *re* anæsthetic responsibility, or indeed concerning anything else. I was once a witness in a case in the Probate Court, which well illustrates this startling truth.

An old lady, who had been an inmate of a mental institution, had been discharged as cured, and was living with her two daughters. On a certain Monday she lost her memory while out for a walk, was found, and brought home. During the days that followed she showed unmistakable signs of recurrent mental instability. On the Friday night she said she felt very ill, was sure she was going to die, and insisted on making her will. She expressed her wishes to her daughters, one of whom put them in writing, and the old lady signed in their presence. The daughters witnessed her signature, being the sole legatees, there was no attestation clause. Early next morning I was called in, the patient was in acute mania, she was certified and taken to the County Asylum, where she shortly afterwards died. The will was disputed by her only son, who lived in a distant part of England. Now this document offends every canon of the law as defined and expounded in any book of medical jurisprudence of which I am aware. The judge pronounced in favour of the will. Personally I thought, and still think,

that this was a shockingly bad judgment, but the various legal luminaries who graced the court precincts appeared to be satisfied. There was no appeal.

The fact is that the law is not bound by precedent. Each case is judged on its own merits, and these include *inter alia* the time era, the geographical situation, established custom, and the individuals concerned. If this were not so there would be no point in going to law at all. Any competent lawyer could predict with practical certainty what must be the result of a given case. As it is the law is a lottery, and anyone who takes a case to court is gambling on his chances, however sound these may appear to him. One learned judge at least, on having precedent quoted at him by counsel, remonstrated, and added that he could not consider himself bound by the bad judgments of his predecessors.

Mr Fullerton remarks that in the past the surgeon was held to be responsible for everything. Rightly so, for in the past he was the sole director of the operation, he chose the room and had it dismantled, he prepared and sterilised his dressings and instruments, and he employed his nurses and assistants. "*Tempora mutantur, et nos in illis mutamur*" No one, I imagine, would accuse the law of marching with the times, but it does move—perhaps I should rather say crawl—after them. Nowadays a surgical operation is an example of team work, each member of the team having his or her own separate responsibility, but the law has not yet discovered this fact.

We still read from time to time of a surgeon being sued for damages, sometimes successfully, for having left a swab in the abdomen, or elsewhere. Now at the present day in normal circumstances the surgeon does not always choose his theatre, he certainly has nothing to do with its administration—the lighting, furnishing, supply of instruments and their upkeep, he does not employ the nursing staff, and as regards the swabs these are not made under his direction, he does not order their size, shape nor composition, nor is he necessarily aware of the number of such contained in each packet. All these details, together with the counting of the swabs before and after use, form part of the duties of the theatre sister and nurses. Under modern conditions of

operative surgery, no surgeon can be expected, neither is it the custom for him to count the swabs, if before closing the wound he has enquired of the nursing staff whether the count is correct, and has received an affirmative reply, he has done all that can be reasonably expected of him. An adverse decision delivered against a surgeon in these circumstances is a monstrous injustice, whatever may have been the merits of such a case fifty years ago. I am still living in hopes that some day a learned judge will acquaint himself as to what really does take place during a surgical operation, and put an end to a form of litigation which is only commenced with the intent of extracting money from a presumed wealthy surgeon, or more accurately from his defence union, which, like all insurance companies, is looked upon as fair game by the unscrupulous.

The editorial comment on Mr Fullerton's letter assumes that the surgeon is responsible for the selection of a competent anæsthetist, and that subsequently everything concerning the anæsthetic is the responsibility of the anæsthetist. If the first part of this assumption should indeed become the law, or even the general custom, it would be a good day for the public as well as for the anæsthetist. There is nothing to be gained by looking upon this matter from the viewpoint of the ostrich whatever may be the practice in London within a mile or two radius of Hyde Park and in a few large provincial cities, a very large proportion of operators up and down the country, whether actually surgeons or not, do not take any steps to choose a competent anæsthetist, on the contrary they only too frequently deliberately avoid such a choice in order to profit financially. Where the operator is himself in general practice, one of his partners gives the anæsthetic in order to "keep the fee in the firm." Where the case has been sent to the surgeon by a general practitioner, the latter acts as the anæsthetist, whether he has any knowledge of the art or not, because the surgeon is well aware that if he offers any protest no more surgical cases will be referred to him from that quarter. I know a surgeon who found himself so hampered by the inadequate anæsthesia habitually served up to him by one practitioner, that he was compelled in self-defence to invent an indication for spinal

analgesia, which he administered himself, at every possible opportunity I knew of a surgeon, of such seniority that he should have known better, whose practice was to employ as his anæsthetist the latest newcomer to the town, in order to get his surgical connection, after "running" him for a few months he would cast him aside for the next arrival. In all the foregoing instances either the surgeon, or the patient's own doctor, or both in collusion have plainly failed to provide the reasonably competent service to which the patient is entitled, they are therefore liable to an action for damages for negligence if any accident or complication arises as a result of the anæsthetic.

There is always the chance that an alert coroner, faced with an anæsthetic fatality, may shatter the complacency of the inexperienced anæsthetist. Where in the course of his inquest it transpires that a wealthy patient in a position to afford expert attention, has arranged to pay a large fee for operation, has entered a private nursing home, and has had foisted upon him an unskilled anæsthetist with disastrous results, when all the time a full-time anæsthetist was available for the asking, the coroner may begin to toy with a verdict of manslaughter. The inevitable result of the mere suggestion will be an action for damages in another court. This will awaken the public at once, for nothing is better calculated to arouse them than the possibility of a fresh field for profitable litigation.

In the case where a surgeon habitually calls in an anæsthetist, and after the operation is ended the former slips a couple of guineas into his hand with the intimation that that is his fee, it appears that the anæsthetist might be able to plead that he was an agent acting for his principal, the surgeon, who is responsible for his torts. He has certainly had no opportunity of effecting any contract with the patient himself, it is customary and proper for a patient to pay his specialist's fees direct, and it is probable that in many instances the anæsthetist considers that the fee paid is grossly inadequate for the services rendered, and would himself have contracted with the patient on a higher scale.

There is an incongruity arising from the existence of the Medical Register, which can hardly have been intended or

foreseen The assumption that once a man has obtained a medical qualification entitling him to registration, he is thereby capable of undertaking work in any and every branch of medicine or surgery is an absurdity which invites challenge There is a vast difference between the good-all-round practitioner and the "universal specialist" whom most of us have met There has been a good deal of opposition expressed of late in the correspondence columns of the medical press to the various specialist diplomas which have been created in recent years, originating, I suspect, from those who would find considerable difficulty in obtaining them, protesting the while the superior virtue of "experience", a term which might on occasion be defined as "doing the same job consistently badly for thirty odd years or so" Actually they present no great obstacle to those for whom they are intended, the standard required is not of a very high order, but they do demand some special knowledge, together with a minimal period of practice in the particular subject selected, moreover, the cost is well within the reach of the quite humble pocket I should have thought these diplomas to be singularly free from objection, and a godsend alike to those who desire to embark on a specialist's career, bodies responsible for the filling of vacant medical appointments, the public, and even the law

There is another group of persons for whom I am convinced there awaits a rude awakening in the not very distant future, I refer to the management committees of institutions Time and again these bodies invite applications for staff vacancies, select a short list for interview, hold a farcical meeting, and then proceed to "elect" a local aspirant with very doubtful credentials They feel secure in their belief that the medical staff are alone responsible for any claims for negligence which may arise out of treatment This may have been very well when hospitals really were charities, but a new factor has arisen In most cases hospitals are now receiving direct payments from their patients, in many the staff are themselves paid, and those who pay have a right to expect skilled attention What of the man who is appointed anæsthetist merely to get a footing on the staff, who is content to "do anæsthetics" until a vacancy occurs for a surgeon or

physician? A little time back a county council invited applications for a paid anæsthetist. They had present at interview three excellent candidates, one of whom had many years of experience and possessed the diploma, and they selected the fourth, whose sole qualification was the L S A, obtained the previous year.

A certain hospital, to which is attached a medical school, as recently as twelve months ago had not a single whole-time anæsthetist, all were general practitioners "doing" anæsthetics. To their credit the lay members of the committee were far from satisfied with this condition of affairs, but any attempt to remedy it was met with persistent opposition from the medical members, who told them it would not be possible for a whole-time anæsthetist to make a living. And this with a population of half a million!

Finally, I feel sure that not only would a fully trained and qualified anæsthetist (as distinct from his "pseudo" rival) be held entirely responsible in law for everything immediately concerning the anæsthetic, but, and this is equally important, he would find himself fully protected by the law, and no surgeon need have any misgivings regarding the outcome of a charge of negligence in the administration of an anæsthetic if made against himself in such circumstances.

PREGNANCY—A CONTRA-INDICATION TO SPINAL ANALGESIA

By F BARNETT MALLINSON, M R C S , L R C P , D A ,
R C P S

*Anæsthetist Dental Department London Hospital, Hon
Anæsthetist Woolwich Memorial Hospital, St John's Hos-
pital, S E , and Eltham Hospital, S E*

THE article published in the *British Journal of Anæsthesia* for April 1938, by Dr Van Der Post describing three cases in which spinal analgesia was administered with unfortunate results, and the subsequent articles which appeared in reply in the July number of the same *Journal*, have prompted the author of these notes to join in the defence of what is considered by many to be an important and sound advance in the technique of spinal analgesia in modern times, namely, the Etherington-Wilson technique. At the same time it is noted that Dr Van Der Post invites criticism and advice.

So far as the present discussion is concerned, the possibility that the state of pregnancy in itself constitutes a contra-indication to the employment of spinal analgesia, does not seem to have received due consideration. It is the author's intention to try and show that this may well be so.

An examination of the literature on the subject reveals that a number of workers have come to the conclusion that there is some peculiarity in the physiology of the pregnant woman which renders her unsuitable for the induction of spinal analgesia. De Lee¹ quotes Kronig as saying that "spinal analgesia is dangerous in pregnancy and labour." Winter and Halban² also mention the danger of spinal analgesia in women who are pregnant or in labour. Again, Kidd³ gives it as his opinion that there is an increased risk in the use of spinal methods in advanced pregnancy, and so do many others.

The question is not merely one of an increased splanchnic vascular bed and its dilatation under the influence of spinal nerve paralysis, nor yet one of the presence of a large abdo-

minal tumour and the results on the intra-abdominal pressure of its sudden release. No mention seems to have been made of there being any danger to be apprehended from using a spinal method for ovarian cystectomy or the removal of large fibroid uteri. Both these factors do apply to cases 1 and 2 in Dr. Van Der Post's article, but there are further factors which do not yet appear to have been taken into consideration in discussing these cases, concerned with the physiological make-up of the woman who has conceived. Voron⁴ alludes to the "Special Bulbar Sensibility" of the cytic woman.

If one now turns to a consideration of some facts concerning the physiology of pregnancy, it will be found to be generally agreed^{5,6,7} that amongst other physiological aberrations of this condition the blood and the central nervous system both show variations from the normal.

Blood It is stated with regard to the chemistry of this tissue that it has a diminished CO_2 combining power in pregnancy and that therefore there is an increase of CO_2 in the blood with a lowered CO_2 alveolar tension, i.e., a certain degree of acidosis. In addition Williams⁶ describes the discovery of various substances present in the blood in pregnancy ranging from increased cholestrin and anterior pituitary substances to histamine.

Central nervous system With regard to this system it is stated, and indeed it is an easily observed phenomenon, that the reflex irritability is markedly increased. This is well shown by the bladder as early as the second month of pregnancy.

Consideration may now be given to the alterations in physiology found in the organism under the influence of spinal analgesia. Under these conditions vaso-constrictor, as well as motor and sensory, paralysis occurs in relation to the segments affected.

Co Tur⁸ states that if an excess of CO_2 is superimposed on the vaso-constrictor paralysis, the residual tone of the vessels is lost and the blood-pressure may reach zero. It is further shown by Seevers and Waters⁹ and by Heymans and others¹⁰ that in animals under spinal block excess of CO_2 lowers the blood-pressure by peripheral dilatation. Again Saklad¹¹

states that a condition of decreased CO_2 combining power of the blood is associated with a shock-like state. In parentheses it can be argued that CO_2 is thus not a good respiratory stimulant for use under spinal block. This argument is borne out by the fact that a marked increase in pulse-rate can frequently be observed if CO_2 be administered during the course of an operation under spinal analgesia.

Now in pregnancy the CO_2 combining power of the blood is diminished and therefore the CO_2 content of the blood is increased. Thus one would expect a greater fall of blood-pressure to take place as a result of the administration of spinal analgesia in the pregnant than in the nonpregnant patient. These considerations are certainly suggestive of the likelihood of trouble arising from the combination of pregnancy and spinal analgesia.

Assuming, at any rate in the vago-tonic type of patient, a preponderance of vagal action over sympathetic, the likelihood of cardiac depression arising as a result of increased vagal tone produced through vagal overaction seems very real in patients whose reflex irritability is seen to be above normal. Such cardiac depression might also contribute a great deal towards the lowering of the blood-pressure, which is to a great extent one of the bugbears of spinal technique.

Lastly, it might well be that some or other of the substances described as being present in the blood in increased quantity or only present in the blood in pregnancy, or substances in the blood of pregnant women as yet undiscovered may exert some effect perhaps in the direction of increased vaso-motor instability rendering any method of spinal block a more hazardous proceeding than general anaesthesia in the pregnant patient.

In cases 1 and 2 of Dr Van Der Post's series, as has been already pointed out,¹² the patients were not good risks for any form of anaesthesia. There seems to be little doubt that whatever method had been employed these patients would have given rise to a good deal of anxiety. The first patient already had a low blood-pressure and "looked ill". The second patient must have been considerably exhausted to have come to Cæsarean section after 15 hours in labour. If, then, one adds the theoretical considerations put forward

above, which one would expect to result in further lowering of blood-pressure were spinal technique employed, then the likelihood of trouble arising becomes pretty well a certainty

One cannot see, then, that the actual technique should be called in question but rather the application of any spinal technique to unsuitable cases

The dosage employed, 10 c c percaïne 1/1500 is definitely on the low side, and the sitting-up times the shortest that one would expect to be sufficient for the purpose When a "high" spinal is called for, such as is commonly employed in radical gastric surgery, it is a usual practice to use 15 to 16 c c of percaïne and a total sitting-up time of 65 seconds, 20 seconds for injection plus a further 45 seconds The author has used these figures on many occasions preceded by a pentothal induction in addition as described by Jarman¹³ It can be truthfully said that this technique has not as yet given any real cause for anxiety, but at the same time it is emphasised that it has not been employed for operations on the pregnant patient, because one has felt convinced that a contra-indication definitely does exist

The following case report may perhaps add a little more force to the argument against the employment of spinal technique where the condition of pregnancy exists This case recently came under the author's notice, and the following details were made available

CASE REPORT

Female Aged 30 years

History Complained of abdominal pain Six weeks amenorrhœa, followed by continuous slight bleeding for the last 10 days One day ago, heavy loss of blood *per vaginam* Two hours ago, onset of severe right-sided abdominal pain

Past history One child

Condition on examination Temperature 98°F, pulse 80, respiration 24, blood-pressure 140/90, pale, tongue slightly furred *Heart and lungs* nothing abnormal detected *Abdomen* obese, tenderness and muscle-guarding right lower quadrant *P V* tenderness in right fornix

Diagnosis Ruptured ectopic gestation

Anæsthetic Spinal analgesia was decided upon because absolute relaxation was desired owing to the very fat belly

Method Percaïne 1/1500, 14 c c, third space *Sat up* for

40 seconds Ephedrine, gr $1\frac{1}{2}$ injected $N_2O-O_2(O_2=35\%)$
administered

Collapse About 45 minutes after the injection had been completed, respiration became depressed, pallor and slight cyanosis developed, the patient being in the Trendelenburg position, and the surgeon being occupied in suturing the peritoneum. Shortly afterwards respiration became imperceptible and then ceased.

Measures taken (1) Lobeline subcutaneously, artificial respiration, CO_2 . (2) Patient de-Trendelenburged. This was done apparently because it was considered that blood, etc., pressing on the diaphragm was helping to embarrass respiration. (3) Cardiac puncture. (4) Adrenaline into the heart. (5) Cardiac massage.

No response was forthcoming to any of these measures and the patient was eventually given up as dead after a prolonged effort at cardiac massage and artificial respiration.

Some of the measures taken to resuscitate the patient in the above case may be open to criticism. Subcutaneous injection was probably only a slight waste of time. De-Trendelenburging the patient might well be considered inadvisable, being contrary to orthodox practice in such emergencies, also the sudden change of position could be calculated upon to upset still further the already damaged vaso-motor centre. These considerations, however, have nothing to do with the original causes of the collapse.

This patient's general condition appeared in the circumstances to be definitely better than the average, as also was the general condition of case 3 in Dr Van Der Post's series. Temperature 98, pulse 80, and blood-pressure on the high side, the latter being perhaps beneficial from the point of view of inducing spinal analgesia. In addition the heart and lungs were normal. There would seem, therefore, to have been no reason to expect trouble. The dosage was above that used in Dr Van Der Post's series but well within the limit of safety, the sitting-up time (40 seconds) was not excessive. Yet the collapse occurred, resuscitation failed, and so the patient died adding one more case to the list of fatalities under spinal analgesia in pregnant women.

SUMMARY

(1) Three cases of serious collapse during spinal analgesia described by Dr Van Der Post, two of them ending fatally, are considered.

(2) It is pointed out that in each case the patient was in the pregnant state

(3) The opinion of a number of workers is quoted tending to show that their experience of spinal methods of analgesia in pregnancy has not been happy or fortunate

(4) The suggestion is put forward that pregnant patients may be "Rachi-sensitive" to spinal analgesia, and an attempt is made to produce a plausible theory to account for this on a physiological basis

(5) This theory concerns itself primarily with the altered blood-chemistry of pregnant women, and secondarily with the abnormal state of their central nervous systems

(6) Two at least of Dr Van Der Post's cases were of a nature likely to give rise to anxiety whatever method of anæsthesia was adopted, and assuming the theory mentioned in (4) and (5) to be a real factor, trouble in these two cases could confidently be expected, while in case 3 it could be forecast as probable

(7) A further example of death under spinal analgesia coming within the author's scope of observation and having the common factor of the presence of the pregnant state is described and reviewed

(8) In the author's opinion, therefore, there is some reason for suggesting that pregnancy constitutes a contra-indication to the employment of spinal methods of analgesia

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THE DIPLOMA IN ANÆSTHETICS

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- J A W Robinson, M B , B S (Lond), King's College and Guy's
Hospital
S G Shippard, L R C P , M R C S , King's College
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ANÆSTHESIA IN CARDIAC SURGERY

By J K HASLER, M B , D A

AT one of the sessions of the surgical section at these meetings* Mr Laurence O'Shaughnessy is to read a paper on "Surgery of the heart" As I have had the opportunity of giving the anæsthetics for those cardiac operations which Mr O'Shaughnessy has himself performed, I have thought it appropriate to give a brief account of the cases with special reference to the anæsthesia employed Nearly all the cases to which I am referring were operated on at the L C C cardio-vascular clinic at the Lambeth Hospital I am indebted to Sir Frederick Menzies for permission to give an account of our experiences at that clinic

Until recently operations on or about the heart were, at any rate in this country, of infrequent occurrence They were usually undertaken as heroic treatment for a rather desperate condition The operations which have been performed include the removal of foreign bodies, such as bits of shrapnel, from the myocardium, the repair of stab wounds, and on occasions attempts have been made to deal with pulmonary emboli During the past few years attempts have been made to relieve the ischæmia produced by occlusion of the coronary arteries by attaching grafts of intercostal muscle or of omentum to the heart muscle itself It is about these last cases that I wish to speak The majority of the cases were suffering from cardiac ischæmia associated with angina pectoris and had had at least one severe attack of that malady Their disease had placed them under the care of a physician and when it appeared evident that medical treatment offered little chance of improvement they were sent to the surgeon to see if a surgical operation could give relief While some of the patients were able to lead a

* An occasional paper read at the B M A meeting, Plymouth

normal though quiet existence others had had to give up work and a few were more or less bedridden. Some of them experienced cardiac pain on slight exertion or when cold, and in others it occurred with emotion such as was produced by reading a thrilling novel or watching an exciting film. For these patients it was decided to perform the operation of cardio-omentopexy whereby an additional blood-supply could be provided for the heart muscle by grafting the great omentum on to the myocardium. Needless to say all the patients who were operated on would be regarded as bad risks for a surgical operation and most of them were middle-aged or elderly. I thought it desirable that these patients should, by adequate premedication, be spared the mental anxiety which precedes an operation, and I therefore endeavoured to bring them to the operating theatre either asleep or else so drowsy that the memory of the event would be very hazy. On the evening before operation a sleeping draught was administered to ensure a good night's rest. This was usually chloral and bromide. On the following day, one hour and a half before the scheduled time of operation, each patient was given a hypodermic injection of omnopon, either gr $\frac{1}{3}$ or gr $\frac{2}{3}$ and scopolamine gr $\frac{1}{150}$. This was followed in half an hour by a capsule of nembutal gr $1\frac{1}{2}$ given by mouth, and if at the end of another half an hour the patient was not asleep, or, at any rate, very drowsy, a second capsule of nembutal was given. This is a form of premedication which I have used frequently during the past four or five years and which has proved very satisfactory, especially before spinal anæsthesia. All the cardiac patients had this form of premedication and many of them arrived at the theatre quite asleep. Of those who were not quite asleep the majority had little or no recollection of the event afterwards. General anæsthesia was induced with an ethyl-chloride ether sequence on an open mask and when surgical anæsthesia was reached the mask was removed and replaced by a face-piece connected to a Tiegel-Henle apparatus. This piece of apparatus is German in origin, and although it looks rather large and complicated is in reality quite simple. A glass vessel which contains ether is fixed at the top of the apparatus and from this ether drops into a chamber through

which a stream of oxygen can be passed. The mixture of ether and oxygen passes along flexible tubing to the face-piece where it is inhaled by the patient. A rebreathing bag is inserted between the ether chamber and the face-piece. The expired gases are conveyed away from the patient by a second piece of flexible tubing which runs from the face-piece to a straight metal tube which dips into a glass jar containing water. The lower that this metal tube dips into the water the greater the column of water that has to be displaced by the expired gases in order to escape. By this means the pressure inside the apparatus can be increased and a positive pressure produced inside the patient's lungs. It is, of course, essential that the face-piece should fit accurately or a leak will occur. I have usually employed a Clausen's harness to keep the face-piece in position, and even in edentulous patients who have hollow cheeks I have managed to produce an accurate fit by inserting an airway. I have not found it necessary to insert an endotracheal tube though several observers have expressed surprise that a positive pressure can be produced without its use.

In the majority of cases the transpleural route was chosen for the operation. Briefly stated the procedure was as follows. Having gone through the chest wall the surgeon opened the pleural cavity and temporarily paralysed the left side of the diaphragm by crushing the left phrenic nerve with a pair of artery forceps. An incision was then made in the diaphragm and the great omentum drawn up through it. The omentum was then anchored to the slit in the diaphragm and the free end was passed through an incision in the pericardium. It was then either attached to the left ventricle by means of a stitch or anchored to the slit in the pericardium so that its free end was in contact with the ventricle and in a position to become adherent. After this the wound in the chest wall was closed.

At the commencement of the operation it was unnecessary to use any positive pressure while giving the anæsthetic and the metal tube on the apparatus was adjusted so that its opening was just below the surface of the water. As soon as the pleural cavity was opened the left lung collapsed and positive pressure was produced by lowering the metal tube

into the water to a depth of 5 or 6 cm. It was usually kept in this position until the main part of the operation was completed. During the closing of the chest wall positive pressure was still further increased by lowering the metal tube to a depth of about 8 or 10 cm. and it was kept in this position until the end of the operation. It will thus be seen that during the whole of the operation the patients have been supplied with the maximum quantity of oxygen. Even while the left pleural cavity has been open the left lung has been prevented from remaining collapsed by means of positive pressure. One fact with regard to the use of positive pressure is of interest. It has been found when operating on animals that during the time that a positive pressure is employed small quantities of gas may pass down the oesophagus and distend the stomach. It might be thought that a distended stomach would interfere with the finding of the omentum and on a few occasions the surgeon has asked me to compress the oesophagus by applying digital pressure to the trachea until the omentum has been brought up. Only on one occasion has a distended stomach proved a handicap and the face-piece had to be removed for a few minutes while a stomach tube was passed. How much of the distention may have been due to the passage of oxygen down the oesophagus I am unable to say. In most cases, however, no difficulty has been experienced in finding the omentum nor has the stomach been unduly distended.

After operation the patients are kept at rest for a period of two or three months to enable the graft to become properly established before any exercise is taken. It has been satisfactory to see how remarkably well the patients in this series have stood the operation. This I attribute very largely to the fact that during the period of anæsthesia they have been liberally supplied with oxygen. They have, to all intents and purposes, had their anæsthetic while in the equivalent of an oxygen tent. It has not been necessary to use a great deal of ether for these patients. This is partly due to the fact that some rebreathing takes place inside a closed apparatus and partly because these patients have had liberal premedication and deep anæsthesia is not required

c

On some occasions the amount of ether used has been at the rate of $1\frac{1}{2}$ to 2 oz per hour. From the anæsthetic point of view none of these patients has caused the least anxiety while on the operating table and it is usual for them to leave the theatre with a good colour and a steady regular pulse.

Patients suffering from angina pectoris are liable to die a sudden death from coronary thrombosis at any time, and of the patients who were sent up to the clinic with a view to having operative treatment several died in this way between the time when they were seen by the surgeon and the day when it was proposed to operate. There have also been two deaths from coronary thrombosis during the week following operation before the extra blood-supply from the omental graft could become established. One patient died ten days after operation as the result of hæmorrhage from an old duodenal ulcer and one patient died a week later from pneumonia, this being the only case in which the anæsthetic could in any way be held responsible for death.

In addition to the heart cases already mentioned I have also used this form of anæsthesia for various other operations on the chest such as thoracoplasties, exploratory thoracotomies for carcinoma of the lung, for a diaphragmatic hernia, and for a few other cardiac cases which are not in the angina group.

In this somewhat brief paper I have tried to show that for cardiac operations it is possible to administer anæsthesia without unduly endangering the life of the patient. Two things I regard as important in this work. First, the giving of careful and adequate premedication, and second, the administration of a liberal supply of oxygen.

ABSTRACTS

"Tonsillectomy under intravenous anæsthesia in children"
 K HUTCHINSON, H S MITCHELL and H McHUGH in
Canadian Med Assoc Journ, September 1938, p 237

THE authors point out the frequency with which throat operations are required for children afflicted with tubercular and other respiratory disease. Not entirely satisfied with the operative facility provided by avertin and nitrous oxide the authors have employed intravenous anæsthesia with evipan or pentothal. The latter they find too formidable a respiratory depressant. They use morphia hypodermically beforehand and find that this improves results. They do not find that difficulty regarding entrance to a vein is any obstacle even in children under eight years of age. The cough reflex was not always abolished, and when so returned soon after operation.

"Vinethene in dentistry" H FELDMAN and S CARTIN in
Dental Outlook, November 24th, 1937, p 504

THE authors bring out well the great value of divinyl ether in expert hands. The times both of induction and of available anæsthesia compare favourably with those of nitrous oxide and safety and comfortable recovery are as well assured. A simple drop method of administration is used, the mask being of eight thicknesses of gauze and being capable of remaining over the nose during operation if this is wanted. Owing to the extreme volatility of vinethene it would appear that large amounts must be used up even for short operations.

A new apparatus for administering vinethene is described in *Current researches in anæsthesia* for July-August, p 91, and another on p 239.

"*Basal narcosis for tetanus*" P T O FARRELL in *Brit Med Journ*, August 13th, p 348

SEVERAL instances have been recorded since 1927 of the successful use of avertin in the treatment of tetanus. The present case is one in which intravenous nembutal was employed with equally satisfactory results. The patient was a robust man of 28 years of age who was admitted to hospital showing the classical signs and symptoms of generalised tetanus. For the first three days continuous narcosis was kept up by intravenous injection, at intervals, of $7\frac{1}{2}$ grains of nembutal during which time ten pints of glucose saline were given by continuous intravenous drip. From the fourth day on the nembutal was given by the mouth in $4\frac{1}{2}$ gr doses allowing intermittent narcosis so that food could be given. Over the period of ten days 75 grains of nembutal were given. The patient recovered completely.

"*Helium in anæsthesia*" W S SYKES and R C LAWRENCE in *Brit Med Journ*, August 27th, p 448

THE value of helium as an inert gas lighter than nitrogen has lately received attention from anæsthetists. The authors of the above-named article conducted personal experiments showing the value of the gas when breathing was carried out through a restricted tube. The times for which they could inspire a helium oxygen mixture were always greater than those possible when they inspired ordinary air regenerated, a nitrous oxygen mixture. The absorption of helium from the alveoli into the blood is exceptionally slow, and also exceptional, as the authors point out, is its facility in passing through narrow apertures. They conclude that an atmosphere of helium and oxygen is about twice as easy to breathe as ordinary air and the mixture should be of great value in cases of respiratory obstruction or in cases in which it is essential to economise muscular effort. Also helium helps to avoid post-operative collapse of lung because of its low solubility.

"*Sodium thio-ethyl anæsthesia*" S CULLEN and E A ROVENSTINE in *Current Researches*, July-August, p 201

A CAREFUL investigation has been carried out firstly on

animals and then on 100 human subjects. The drug sodium thio-ethylamyl is the sulphur homologue of sodium amytal. The authors exercised due caution in selection of patients and except in one instance no long anaesthesia was attempted. Results were good but apparently not superior to those gained from the more usual barbiturates, but the true position of the new drug cannot be stated without fuller trial. Respiratory irregularities were common during anaesthesia but may be prevented by the preliminary use of atropine. The authors remark that for severe depression or arrest intubation is necessary. One patient developed numerous urticarial wheals during the administration.

"The influence of certain basal narcotics on the haemoglobin saturation" D. CORDIER and P. SOULIE in *Anaesthesia et Analgesie*, June 1938, p. 285

THE authors have carried out a number of experiments on dogs. They arrived at the conclusion that even in the excessive doses employed for experiment neither evipan, rectanol nor narcosol had any sensible effect on the affinity of the red corpuscles for oxygen. There was also only slight alteration in the sum of ethero-soluble acid in the arterial blood.

In the same journal, on page 293, L. Dautrebande starts a long article on recent progress in anaesthesia. Little new ground is broken but there is a useful paragraph dealing with the advantages of oxygen inhalation during and after anaesthesia. The author points out that study of the oxy-haemoglobin of the venous blood shows that all anaesthetics except cyclopropane are followed unless the inhalation has been very short by a fall in the haemoglobin oxygen saturation during the days following anaesthesia. Moreover, the hepatic insufficiency which is well known to follow inhalation of potent anaesthetics has been proved to be lessened or prevented by the avoidance of any diminution of the patient's oxygen saturation during anaesthesia. This can only be secured by judicious use of oxygen with the anaesthetic.

REVIEW

"Hypnosis, its meaning and practice" ERIC CUDDON,
M A Bell & Sons, London, 169 pages, price 3s 6d

ANÆSTHETISTS are naturally interested in hypnosis, a state not far removed from that with which they are daily familiar. Our readers are likely, therefore, to enjoy this little work which gives a good general idea of hypnotism and is freely embellished with quotations from those who have done most to establish its reputation and practice. The author perhaps flogs a dead horse too often in his protestations that hypnotism is not humbug, everyone with any scientific and medical training knows the basic facts and sees no direct association of hypnotism with spiritualism or even of the former with telepathy or with clairvoyance. Perhaps the most interesting pages are those which deal with post hypnotic suggestion, and many illuminating experiments are quoted in detail. The author shows how difficult it is, outside the pages of fiction, for hypnotism to be put to criminal or felonious purposes. On the other hand, he suggests, without going into practical points, the various directions in which hypnotism may usefully serve a medical man's objectives. He does not point out a truth which is probably familiar to our readers in that successful anæsthetists rarely pass a day without employing the useful aid of "suggestion." We confess to surprise that in a book which otherwise displays much sound judgment and knowledge the professional medium and his "spirit" communications should be taken seriously. The author does not think it unreasonable to suppose that in mediumistic trance the inner mind of the medium may receive and interpret "impressions, the source of which is the spirit attempting to communicate." Here we would definitely draw swords with him. It is most unreasonable, since anything for which there is more dubious evidence than the existence of spirits, still more their presence in mediums, we believe it would be hard to produce





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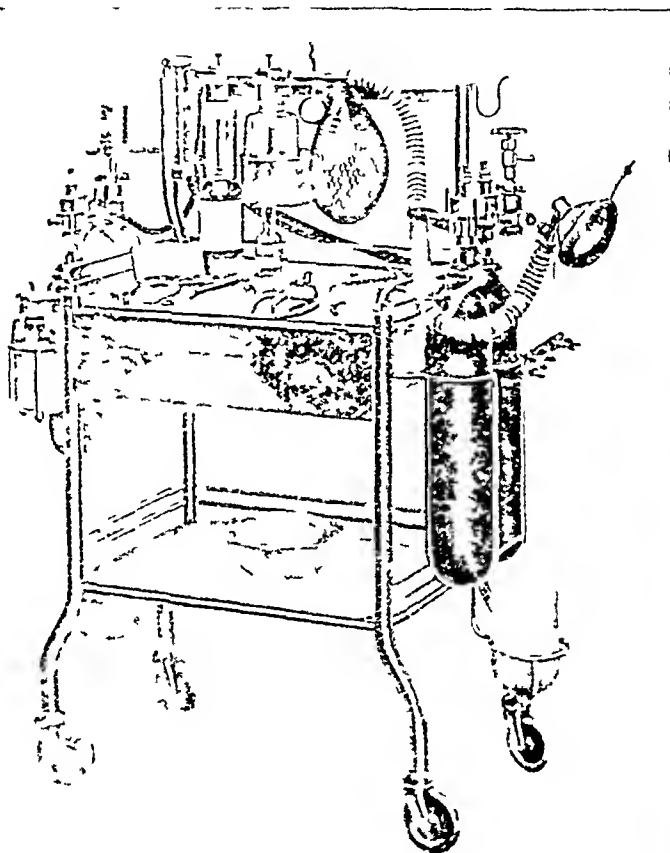
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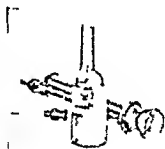
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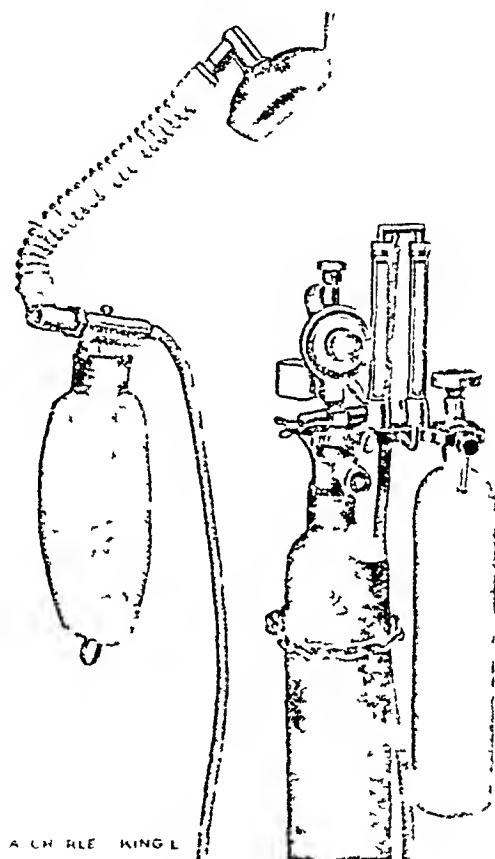
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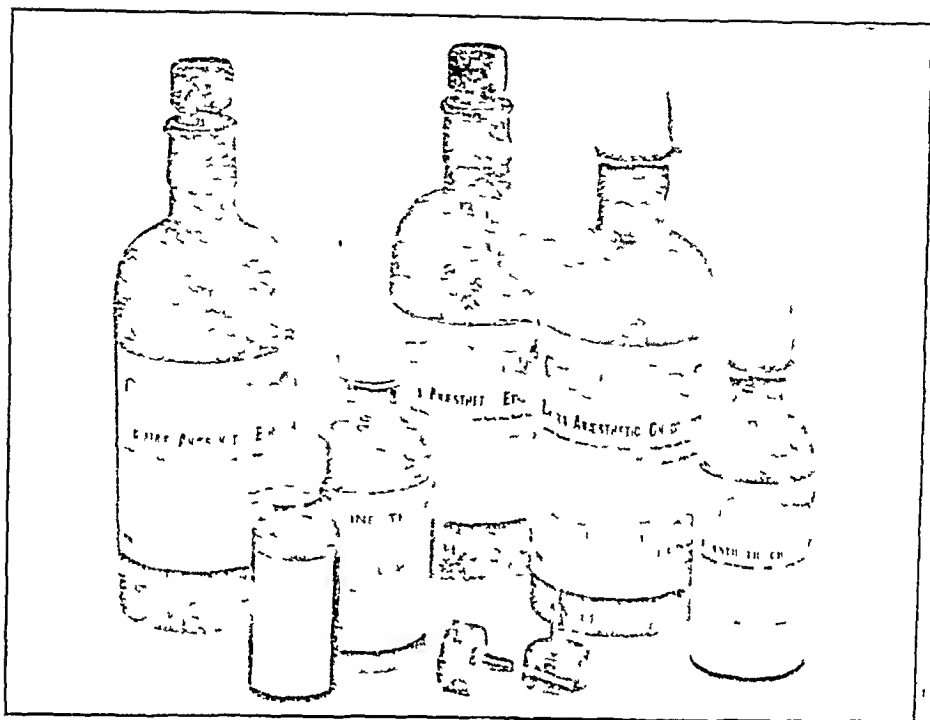
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June 1936

ETHER CONVULSIONS

By A S HOSEASON, M D , M R C P ,

Laking-Dakin Research Fellow, St George's Hospital

THE first references to ether convulsions appear in the *Lancet* and *British Medical Journal* in 1927, and, by a curious coincidence, in issues of the same week, they were by S R Wilson and K B Pinson respectively. Since then, many articles and letters have appeared, but it must be admitted that there has emerged no definite clue as to aetiology or non-empirical treatment. The various conflicting theories are well-known and the literature needs no exhaustive survey or analysis. A complete summary of the position has appeared recently (Woolmer and Taylor). The only factor common to all cases, with one exception, is the ether. Septic toxæmia almost always is present, and frequently excessive heat, the cases occurring more often during very hot weather. The patients are usually youthful or young adults. The other circumstances are variable in the extreme, and the treatment, in a condition where the real cause is unknown, of necessity is equally uncertain. Further confusion is caused by the fact that cases may have convulsions during one anæsthetic and yet be quite normal during subsequent anæsthesia, and vice-versa, while the same ether may be used in another case with no ill-effects.

No suggestion is completely satisfactory. Since the ether does not seem to contain in itself the toxic or convulsant agent, it is likely that one must look to the secondary changes known to be produced in the *milieu interieur* by ether anæsthesia in general. The principal changes are biochemical and consist in the lowering of the blood CO₂ tension and a tendency towards alkalosis.

The purpose of this article is to suggest that the cause of the fits is possibly connected with a temporary derangement of calcium metabolism such as can occur when the physico-chemical equilibrium of the blood is altered by ether anæs-

thesia Probably several factors co-operate, including that of idiosyncrasy, or convulsion-diathe-sis, and the present and past health of the patient

It must be mentioned, in the first place, that this conception originated in an empirical observation—a case of prolonged ether convulsions that responded dramatically to an intravenous injection of calcium chloride This was given, without much hope, on the grounds that calcium was “good” for convulsive states The administration of calcium becomes less empirical when one considers the following factors, all of which operate to depress the ionization of calcium in the blood-stream They all combine in their tendency to produce an alkalosis

(1) Ether produces an increased depth of respiration which washes out CO_2 , particularly with ethyl-chloride-ether sequence Clinically, in certain sensitive subjects, hypernoea causes tetany, even when the hypernoea results from physical exercise Tonic spasms (not ether clonus) are sometimes seen in the induction stage, and there may be laryngeal spasm as in tetany, even though the ether vapour is not in an overwhelming concentration These spasms are preceded by a gradually increasing hypernoea

(2) Modern methods of anæsthesia, intubation in particular, tend to lower the alveolar CO_2 tension Although respiration may be very shallow, it is 100 per cent effective as the dead space is by-passed It is found experimentally that a very considerable alkalosis develops with the progress of anæsthesia

(3) Sepsis, particularly of the peritoneal cavity, is often present This is said to depress the level of the serum calcium, but it is difficult to know whether this is a direct result of the sepsis, or (more likely) a by-effect of the vomiting usually associated with abdominal crises This may be compared with “gastric tetany” and the biochemical sequelæ of pyloric and high intestinal obstruction

Younger patients are more subject to these convulsions probably because they have a more labile metabolic balance Not only are abdominal emergencies more common among them, but they are far more easily upset and prone to vomiting than are elderly patients Children vomiting from

some simple gastric disorder may show the most extraordinary ketosis, and here again is the unguessable factor of idiosyncrasy, for while one child may be extremely ketotic, another may be relatively unaffected by the same degree of vomiting

Other factors of importance are the blood changes occurring during mild heat-stroke, aided by the effects of atropine in checking heat and fluid loss. It is also possible that oxygen-lack may play a part, for although the blood may be fully saturated with oxygen, the low CO_2 tension inhibits its dissociation. Nervous tissues are highly sensitive to oxygen-lack, and this may render them unduly susceptible to small changes in their chemical environment. This point is difficult to assess, for while the figures of hæmoglobin dissociation curves clearly emphasize the importance of CO_2 , it is doubtful whether a moderate decrease in CO_2 could have such profound effects on function, although the presence of an alkalosis may considerably intensify them.

Unfortunately, simple estimations of the serum calcium are not very informative, since it is the ionized and physiologically active fraction which determines neuro-muscular irritability, tetany often occurs with a normal serum calcium level, and sometimes the total figure may be well above normal.

On the strength of these considerations, the writer always had at hand a syringe and a preparation of calcium, but has never had the occasion to use them. None the less, the treatment indicated has been of success in the one case in which it has been tried. The writer is greatly indebted to Dr F F Cartwright, of King's College Hospital, for his kind permission to quote the details. The case was that of a woman, aged 31, with a ruptured ectopic gestation. The operation was performed in the Trendelenburg position, and after 30 minutes anæsthesia (semi-open ether) typical convulsions developed. Dr Cartwright, who was aware of the writer's views on the subject, gave an intravenous injection of 10 cubic centimetres of a 10 per cent solution of calcium gluconate (Sandoz) and the convulsions ceased almost immediately. It is interesting to note in this connec-

tion that in pregnancy the serum calcium is progressively lowered, and, in fact, from the point of view of calcium metabolism, pregnancy has been compared with hypoparathyroidism. Calcium gluconate (Sandoz) is recommended as it keeps well (obviously a necessity, considering the rarity of the condition) and, unlike calcium chloride, is non-irritant if there is a small perivenous leakage. The injection should be given fairly slowly, but if a vein cannot be entered, owing to the convulsions, 20 cubic centimetres or more may be given intramuscularly.

The writer can only find one mention in the literature of employment of calcium for ether convulsions (H. J. King). This was a case in which treatment was of no avail, and after some considerable time calcium was injected because of its well-known effect in checking neuro-muscular irritability. After the injection, the patient improved temporarily, but the general condition by now was extremely poor and death followed 58 hours after. It is likely that the hyperpyrexia associated with convulsive states in general, and the tendency to heat-stroke in this state in particular, had produced permanent destruction of cerebral tissue. Woolmer and Taylor have successfully employed evipan, and nembutal was given intravenously in one case by Dickson Wright, after a spinal anæsthetic had produced paralysis up to the costal margin. The effect of evipan and nembutal is probably to cause an overwhelming depression of the neuro-muscular mechanism.

One cannot explain why ether convulsions were unknown before 1926 and any theory can be criticized for this failure. The anæsthetics, operations and patients are all much the same, only the methods have changed. The writer's contribution to the many theories of causation rests on observations on two cases only. It may be that an intravenous injection of any hypertonic solution would check the convulsions, this is a matter for trial and experiment. It is, however, a reasonable and plausible suggestion, it attempts to correlate the many apparently unrelated circumstances of the convulsions, if in any way it illuminates the underlying pathology, then it offers a definite line of rational treatment, as opposed to empiricism and symp-

tomatic treatment, and it seeks to draw attention to a much neglected study—the biochemistry of general anæsthesia

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THE CAUSE AND PREVENTION OF "SO-CALLED" ETHER CONVULSIONS

BY RUPERT VAUGHAN HUDSON, F R C S ,

*Assistant Surgeon, Middlesex Hospital, Consulting Surgeon,
Connaught Hospital, etc*

INTRODUCTION

DURING a surgical procedure sudden death without a warning is not unknown. Death preceded by convulsive movements is still considered a rarity deserving of comment. Up to the present time the reason for these fatalities is imperfectly understood, and the suddenness of the onset, the violence of the movements, and the unexpected termination thrust distress and bewilderment upon all concerned. The fatality is all the more inexplicable to surgeons who have seen many patients withstand identical operations and leave the theatre in safety and without incident. There is, however, a certain grim satisfaction in an unsolved mystery.

FOREWORD

On May 2nd, 1936, Woolmer and Taylor¹ published in the *Lancet* an excellent paper. The authors describe four cases of this condition named "Ether Convulsions", they reviewed the literature and gave a definite plan of campaign should such a case occur in any future surgical practice.

Repeated reference is made to this article without apologies for the following three reasons: it is one of the best papers on the subject written, from private enquiry I find that these particular tragedies are increasing and are not as uncommon as previously thought, and, finally, their valuable suggestions appeared one week too late to enable me to prevent, or try to prevent, a tragedy of a similar nature.

Woolmer and Taylor lay stress upon the fact that many

of these cases not infrequently occur when the temperature of the day is excessively hot. They pay tribute to Dickson Wright's² hypothesis of hyperthermia and comment that heat stroke may play a large part in the etiology of the condition.

The following is a description of two cases of “so-called” ether convulsions which have recently occurred in my own practice.

Case 1 My first was typical, the hot, thundery day, the stuffy theatre, below stairs the anxious parents of a young child, overfat, but, withal, a good little patient, suffering from a virulent retrocæcal, appendicular infection which in a few hours had led to a gangrenous perforation and spreading peritonitis, the dose of atrophine but no morphia, and a quiet, uneventful induction with gas and oxygen followed by ether. No anxiety was felt concerning the anæsthetic, as he had had previously, and without incident, a similar anæsthetic for a tonsillectomy.

No case of intra-abdominal perforation is easy to anæsthetize, this case was not an exception and the abdomen tended to be incompletely relaxed.

During intra-abdominal operations it has been my custom to note the colour of the intestines and the pulsation of their arteries in order to assist me in the assessment of the patient's condition.

In about 15 minutes after the commencement of the induction in this case, and while separating the retrocæcal appendix, I noticed that the intestines became ischæmic and then quite quickly cyanotic, the veins of the mesentery becoming distended. Very soon came a single twitch of the patient's whole body, to be rapidly followed by stronger and more frequent twitchings but not violent enough to embarrass the completion of the appendicectomy. As the local colour had improved I was not unduly alarmed.

After a brief space the body was quieter, but while the peritoneum and abdominal wall were being sutured the twitchings recurred, and, becoming stronger and more violent, necessitated the rapid completion of the operation.

Some five minutes from the commencement of the spasms, and 20 minutes from the induction, I was able to examine the patient, and in so doing saw that the movements were becoming more convulsive in spite of the fact that for some minutes the ether had been discontinued and CO₂ and oxygen substituted.

The patient was a terrifying spectacle. Previously a white, quiet boy, he was now a purplish grey, the eyes widely open, and the pupils large and glaring. Each deep breath was accompanied by a convulsive twitch of the facial, the ocular, and skeletal muscles, so

that he raised himself from the table to arch like a bow from occiput to heel. Here, pausing for a second, he rested in rigid spasm, to drop suddenly with a convulsive wriggle, quivering his head, his eyes, and whole body for all the world as if he were a terrier shaking a rat. Spasm rhythmically followed spasm, pitiful to behold, one helplessly witnessed his seizures and the stronger muscles overcome the weaker.

Continuation of CO_2 and oxygen made little difference to his condition, though, certainly, after a short period, the attacks appeared to be lessening in violence and frequency. Intravenous calcium was given, not without difficulty owing to the size of the vein and the movements of the patient, if only this injection had been evipan instead of calcium the issue might have been different.

Twenty-five minutes after the onset and 40 minutes after the induction, I noticed the extreme heat of the patient's body to the touch and the oppressiveness of the theatre. The axillary temperature was taken, and read 106°F , the theatre was immediately aired, but while commencing to sponge down the patient there was a final convulsive movement, and respiration ceased. All further restorative efforts failed.

INTERLUDE

That night I discussed this case with an anaesthetic colleague. I asked him what I could have done, or, alternatively, what he would have done in a similar emergency. His answer was "Your grandfather controlled tetanic convulsions with chloroform. Chloroform has been used with success in such a case, but the intravenous injection of a barbiturate such as nembutal, evipan, or pentothal is considered a better method."

My reply was that evipan had been suggested as a possibility, but I had discarded it at a time when I mistook a period of quiet due to muscular exhaustion for a period of recovery, and, secondly, had I used evipan with a fatal result the coroner would have justly expressed his grave doubts as to the suitability of such a drug in the hands of one inexperienced with intravenous narcosis at a time when spontaneous recovery was still possible.

It is here one must pause to congratulate James Marr⁹ who independently preceded Woolmer and Taylor in the administration of evipan, and by his skilful boldness saved his patient's life. Woolmer and Taylor, in one of their cases, bravely using the same drug, also caused the con-

vulsions to cease, but their patient died some thirty or forty hours later without recovering consciousness. In spite of their success these pioneers do not state why evipan was used, nor do they suggest the method by which this drug obtained its effect, particularly if, as their paper leads one to believe, their patients were deeply under an anæsthetic. Why deepen an already deep anæsthetic?

Some five weeks after my first case my ripening conviction as to the causation of this phenomenon and the rationale of treatment were put to a severe and rigid test.

Case 2 There was admitted under my care a baby two months old, a twin boy, the only pair of twins in a family in which five other brothers had, singly and at convenient intervals, preceded their arrival. In this patient nor in any other member of the family was there a history of epilepsy. The twin brother had gained weight in a satisfactory manner, but this baby, whose birth weight was five pounds eight ounces, was frequently vomiting and not gaining in weight. He looked very ill, pallid, and thin, there was no evidence of pyloric stenosis, but a large, right, congenital hernia was present. With the institution of two-hourly feeds vomiting was not frequent, but in spite of the intensive feeding six ounces only had been gained in five weeks. The hernia was uncontrollable by a truss, and I considered that the hernia was the cause of the loss of weight and that herniotomy was essential.

On the day of the operation the child weighed six pounds six ounces. Here we have an infant, and a very sick infant at that, but no sepsis was present, it was a cool day and there was no thunder except from the anæsthetist, who indignantly refused to anæsthetize such a patient—therefore, there was no anæsthetist. No atropine or morphia was given, and I was forced to perform the operation under local anæsthesia. About 15 c.c. of a 0.75 per cent novocain with one drop of adrenalin was suffused into the operation area in the usual manner. Without waiting the usual ten minutes, as is my practice, the operation was commenced about four minutes after the completion of the anæsthesia, the patient at this time having glucose water out of a bottle. All went well until a very large sac was opened and the cord was being dissected from the posterior wall of the sac, for in a congenital sac the cord is invaginated into its posterior wall. At this point, some ten minutes after the induction of the anæsthesia, the same signs were noticed as in my first case—momentary ischæmia of the intestines followed by venous congestion. To the surprise of the sister, I asked whether the baby's condition was satisfactory. She looked again, and said “No, he has stopped feeding and does not look at all well.” By this time the cord was nearly free, and imme-

diately the first twitch occurred. The operation was stopped and all Spencer Wells removed. The twitches ceased. One bleeding point lay along the ilio-inguinal nerve and had again to be picked up, as the Spencer Wells gripped the ilio-inguinal nerve the twitches at once recurred. The operation was hurriedly completed, but by now the spasms were violent. Here again was the purplish grey colour, the dilatation of the pupils, the facial and ocular movements, the attempt at opisthotonos, the convulsive wriggle, and the rhythmical repetition. To me it was the unbelievable recurrence of a dreadful nightmare, a nightmare softened only by interest and amazement that such a puny infant could make such powerful and spasmodic movements. As in the first case, CO_2 and oxygen proved useless.

Into which vein would the "modern" inject his evipan? I looked at the fontanel and quickly turned to search for "grandfather's" chloroform, ordering the patient back to the ward. Hurriedly following, I reached my minute objective and applied the anæsthetic so vigorously that the convulsions rapidly ceased. So did the respiratory movements. After one or two squeezes of the chest respiration recommenced and without any return of the convulsions.

In the first week this patient gained eight ounces, and in the second nine ounces. He is at present living, and I hope will continue to do so happily ever after, for, typical of the maternal instinct, the good six did not matter provided the prodigal returned.

The circumstances leading to a pitiful tragedy paint such a vivid picture upon the mind that the repetition of the incidents are instantly recognized. This infant's convulsions were not epileptiform, they were not novocain poisoning, they were what has been termed "ether convulsions," and they were produced without the use of any ether and without the use of any anæsthetist. What is more, they were cured by deep narcosis with chloroform.

PREVIOUS THEORIES AS TO THE CAUSATION OF ETHER CONVULSIONS

The following are theories which have been advanced in the past

Idiosyncrasy (Hadfield and Kemp) ⁴ Congestion of the rolandic area by jugular obstruction (Hewer) ⁵ CO_2 accumulation (Pinson) ⁶ Over-oxygenation (Mennell) ⁷ Sepsis Hyperthermia (Dickson Wright) ²

In the evaluation of these main theories advanced as to the causation of ether convulsions, it is important to remember the following points (1) Not one of these factors is constantly present in all cases, (2) fatalities together with

other alarms and excursions occur commonly to the surgeon and anæsthetist who are youthful in experience, (3) ether may be valueless as an anæsthetic in hot countries owing to its rapid volatilization, (4) a patient with a previously raised temperature might easily raise this temperature several degrees by violent muscular effort in a hot and stuffy theatre, and (5) closed circuit anæsthesia is a difficult anæsthetic to give

It would appear to be more probable that these previously advanced theories are contributory to, or, alternatively, the consequence of imperfect anæsthesia

COMMENTARY

Critical analysis of my own two cases leads me to the conviction that two factors were constant (1) hurry, (2) trauma, the two combining to procure intense stimulation of the patient and resulting in shock

Hurry

Natural anxiety to get a seriously ill patient quickly out of the theatre encouraged me in the first case to continue the operation without giving the anæsthetist time to increase the depth of the anæsthesia when the abdomen was found to be insufficiently relaxed, and led me to proceed in spite of the convulsions. In the second, and for the same reason, the operation was commenced before the local anæsthetic had time to suffuse through the whole operation site

Trauma

In each case, at the time of the onset of the convulsions, a hypersensitive area was being surgically interfered with. In the first the peritoneum and the vessels of the mesentery, in the second the peritoneum and structures of the cord, and again, a little later, the ilio-inguinal nerve trunk

Shock

The causes of primary shock are still imperfectly understood. The individuality of the patient, his physical state, and the quality and quantity of the stimuli all play their

part It is true to say that "one man's stimulus may be another man's shock"

Those experienced in the use of local anæsthesia recognize that stimulation of a field of imperfect anæsthesia will give rise not only to an unbelievably widespread distribution of sensation, but also to a definite grading of symptoms according to the particular structures or area stimulated

Remarkably sensitive are the lining membranes of bone and of cavities, the coats of vessels, and certain sphincters Those who have suffered real pain would agree that pain can so invade the whole being that even the slamming of a door will produce intense exacerbation

Must not a serious lesion or long-continued pain facilitate stimuli to reach a wide field of nervous tissues? Those so-called simple manipulations, that sudden dilatation, are they not potentially intensely painful and shock-provoking?

Manifestation of Shock

How are these "hurts" manifested by the patient? (a) by sudden cessation of movement in which the patient gives up the unequal contest, or (b) by sudden movement in ardent protest at the insult

Those convulsive movements described as ether convulsions, are they not a midway house between death and struggling resentment, and their violence indicative of a surgical trauma which has provoked an intense and generalized excitation of the whole nervous system?

The Rationale of Treatment

Evipan, chloroform, even the continuation of ether, each in their turn, have been recorded as controlling so-called ether convulsions Is it not more reasonable to suppose that these individual drugs produce their result by a common mechanism in that they so deepen narcosis that all reflexes are subdued and complete rest is given to the outraged nervous system?

The Relation of Surgeon and Anæsthetist

A patient is placed under the care of an anæsthetist in order to be protected from the painful ministrations of the surgeon The following statements need contemplation

Occasionally the innate fear of a fatal issue and the consequent imputation of overdosage quite unconsciously prevent the inexperienced anæsthetist from giving a sufficiently protective anæsthetic to a very sick charge

Born of a genuine desire for the improvement in the post-operative comfort of the patient, there is a present-day tendency for anæsthetists to attempt to keep patients undergoing painful and shock-producing operations in a state of amnesia rather than in a state of anæsthesia. Time only will show where this modern trend will mislead us

It should, however, appear evident that amnesia and sometimes light anæsthesia is only fair to a patient when adequate and skilled anoci-association is being used as an adjunct

Let us look to the future extension of valuable methods of anæsthetic research, such as the continuous recording of the blood-pressure and the pulse-rate during surgical procedures, as it is only by this means that these contentions will be proved or disproved

At the termination of a paper it is customary to express thanks to one's colleagues. On two scores this is gratefully admitted. First, that one has been brought up to believe and continues the attempt to ensure that an operation theatre is not a place of dispute between a surgical House of Lords and an anæsthetic House of Commons, but a meeting-ground where surgeon and anæsthetist work together on equal terms for the benefit of a fellow-sufferer. It is trusted, therefore, that the outspoken remarks in this paper will not be interpreted as derogatory in character. And this leads me to the second in thanking my friend and colleague, who, by refusing to give an anæsthetic, enabled me, I hope, to submit that the surgeon shares equally with his anæsthetist in the responsibility for the causation of these phenomena previously named “ether convulsions”

The Treatment of so-called Ether Convulsions

Prevention is the ideal. Obviation of shock by the skilful administration of a sufficiently deep anæsthetic and the gentle handling of sensitive tissue. Should convulsions occur there is no time to be lost. The anæsthetist must

acquaint the surgeon in order that he can immediately cease his manipulations and remove all possible instruments that are provocative of stimuli, meanwhile the anæsthetist proceeds to maintain his airway and by the most rapid means in his power renders the patient as deeply anæsthetized as possible. At present evipan appears the quickest and most suitable drug for this purpose.

CONCLUSION

There is one point not yet explained. Why should the first and fatal case occur in an only child, but the second—possessing six brothers—survive? It is submitted that the first to come to experience can leave behind a direction-post for the salvation of the second and perhaps for others of his fellow-men.

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SEQUELÆ OF ANÆSTHESIA

By GEOFFREY KAYE, M D ,

*Honorary Anæsthetist, Alfred Hospital, and Melbourne
Dental Hospital*

AT the present time especially, anæsthetists are endeavouring to improve the quality of their work by paying attention, not only to the safety of anæsthetics at operation, but also to the elimination of undesirable sequelæ. In assessing these sequelæ, they must depend upon statistics taken either from their own practice, or from the records of public hospitals. Of the latter, the annual reports issued by the anæsthetic department of the State of Wisconsin General Hospital might well serve as a model. Institutional records suffer, however, from various defects. They are frequently incomplete, and they are in any case compiled by a variety of anæsthetists, whose methods and criteria are not necessarily alike. Private records, on the other hand accumulate relatively slowly and need to be taken in conjunction with the wider picture presented by hospital statistics. A good example of the value of personal records as a foundation for the individual anæsthetist's views is given by a recent publication by Lukis.² The writer feels that the publication of personal statistics, if frankly presented and sufficiently "controlled," is desirable as a check upon figures obtained from hospital records.

The present series includes 2,520 administrations of inhalational anæsthetics, personally given or supervised by the writer in the period February 1931 to December 1935. Rather more than half of the administrations were performed in hospital practice, and the rest in private practice. The follow-up of the cases was done in this manner: each patient was visited approximately twenty-four hours after the end of the operation, and further information was secured from the surgeon (or, in hospital practice, the

house surgeon), when next encountered This system, although inferior to that ruling in hospitals with full-time anæsthetic staffs, was the best that could be devised under the circumstances, and probably led to most of the undesirable sequelæ being recorded

The cases were distributed as follows

Anæsthetic	Cases	A-Risk	B-Risk	C-Risk	D-Risk
Ethyl chloride	17	17	—	—	—
Ethyl chloride to open ether	360	267	91	2	—
Open ether	38	21	10	3	4
Open ether*with CO ₂	141	85	49	7	—
Endopharyngeal ether†	394	362	32	—	—
Endotracheal ether†	907	788	104	11	4
Chloroform	21	9	9	2	1
Chloroform to open ether	7	5	2	—	—
Nitrous oxide oxygen	188	48	50	78	12
Nitrous oxide-oxygen with ether supplement	124	23	36	62	3
Endotracheal nitrous oxide-oxygen	85	23	26	22	14
Nitrous oxide-oxygen, nasally, for dentistry	124	115	7	2	—
Ethylene-oxygen	57	13	27	13	4
Ethylene-oxygen, with ether supplement	21	1	12	8	—
Endotracheal ethylene-oxygen	36	19	15	2	—
Total	2520	1796	470	212	42

* Including administrations through Boyle-Davis gag

† By "endotracheal inhalation" through Magill tubes, 481

Fatalities

There has been no fatality "on the table" One fatal case of pulmonary congestion following entry of vomitus into the lungs, was directly attributed to the anæsthetic, and is described in detail below Two cases of fatal respiratory sequelæ might perhaps have been averted by different anæsthetic technique, as will be mentioned later No other post-operative deaths were attributed to the anæsthetic, and no useful purpose would be served by recording such fatali-

ties as those following long-standing intestinal obstruction or operations for the removal of extensive cerebral tumours

Choice of Anæsthetic in View of the Anæsthetic Risk

In Australia the "standard" anæsthetic is ether, and the gaseous anæsthetics are reserved in the main for the less good "anæsthetics risks." This is illustrated by the following table

Anæsthetic employed	Cases	A-Risk	B-Risk	C-Risk	D-Risk
Ether (all techniques)	1847	1528=82.2%	288=15%	23=1.3%	8=0.43%
Gaseous anæsthetics, except minor dentals	511	127=24.8%	166=32.5%	185=36.2%	33=6.4%

The D-risk cases included many patients for cerebral operations who, although often robust, were included in this class owing to the hazardous nature of the operation.

The influence of the anæsthetic risk in determining the choice of anæsthetic may be better appreciated from the following table

Risk	Cases	Received	
		Gas anæsthesia	Received ether
A	1655	127= 7.7%	1528= 92.3%
B	454	166= 36.5%	288= 63.4%
C	208	185= 89.0%	23= 11.0%
D	41	33= 80.5%	18= 19.5%

The sicker patients, then, received gas anæsthesia, but ether was used for those patients who could tolerate it with reasonable safety. The choice of anæsthetic seldom erred in respect of safety at the time of operation, but, as will be shown, occasionally did so in respect of subsequent respiratory sequelæ. Gas anæsthesia in good-risk patients usually implied some special indication such as the presence of coryza, of mild diabetes or of renal dysfunction. Ether, when chosen for "C" and "D"-class patients, was usually made necessary by economic considerations. Chloroform

was rarely used, and only when demanded on technical or economic grounds

Post-Anæsthetic Vomiting

The incidence of vomiting was investigated in 1,295 cases, by inquiry from both the patient and the nursing staff. The results may be tabulated generally as follows

Operation	Ether				Nitrous oxide				N O + ether				C ₂ H ₄				C ₂ H ₄ ether			
	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§	*	†	‡	§
Abdominal	23	46	11	5	6	5	1	—	24	17	3	3	5	3	1	—	11	3	1	—
General surgical	61	123	29	7	87	40	10	7	18	11	5	4	5	3	—	1	1	1	—	—
Nose and throat	134	436	58	9	29	12	—	—	8	7	1	—	12	8	2	—	2	—	1	—
Total	218	605	98	21	117	57	11	7	50	35	9	7	22	14	3	1	14	4	2	—

Code — * No vomiting † Slight ‡ Moderate § Severe vomiting
Slight vomiting was taken to mean that the patient was comfortable within three or four hours of awakening from the anæsthetic. Moderate vomiting implied vomiting prolonged until the evening of the day of operation, and severe vomiting anything in excess of this.

The lesser vomiting after gaseous anæsthetics as compared with ether was very apparent in the above figures. Further, "slight vomiting" after gaseous anæsthetics implied less discomfort than "slight vomiting" after ether, in fact, it often meant only a single vomit immediately on terminating the anæsthetic, so that the patient might imagine that he had not vomited at all.

The superiority of gaseous anæsthetics over ether is well illustrated in the following table, in which the above figures are expressed in percentage form. In compiling it, the ethylene cases were combined with the nitrous oxide, as being too few for separate analysis and as being apparently comparable.

Anæsthetic	Cases	No vomiting	Slight	Moderate	Severe
		%	%	%	%
Ether, all techniques	942	23.1	64.2	10.4	2.2
Gases (summed)	353	57.5	31.2	7.1	4.2
Nitrous oxide, without ether supplement	192	60.9	29.7	5.7	3.6

It appeared from these figures that the superiority of gaseous anæsthetics was manifested in the "no vomiting" and "slight vomiting" classes, and that the ratio of moderately-severe or severe vomiting was much the same whether gas or ether was employed. The incidence of vomiting did not seem to be materially enhanced in those cases where ether supplement was employed with gaseous anæsthetics, the addition to the anæsthetic mixture of an ounce or two of ether, spread over an operation perhaps for two or three hours, could exert no very deleterious effect.

An attempt was then made to investigate the incidence of vomiting in terms of the operation performed. Ideally, the data should be tabulated according to the several operations, in the present series the number of operations was too small for this course to be profitable. The data were accordingly grouped as in the following table.

Operation	Cases	No vomiting	slight	moderate	Severe
(a) <i>Ether Cases</i>		%	%	%	%
Abdominal	85	27.0	54.1	12.9	5.9
General surgical	220	27.7	55.9	13.2	3.2
Ear, nose and throat	637	21.0	68.4	9.1	1.4
(b) <i>Gas Cases</i>					
Abdominal	83	55.4	33.7	7.2	3.6
General surgical	188	56.4	29.2	8.0	6.4
Ear, nose and throat	82	62.2	32.9	4.9	—

The figures indicate the great superiority of gaseous anæsthetics over ether in each operation-group, so far as the milder degrees of vomiting were concerned. The more severe grades had much the same incidence, independently of the anæsthetic used, a fact which may suggest that certain individuals, for biochemical or physical reasons, are prone to severe vomiting whatever anæsthetic they may receive. Certain such patients were encountered in the series. Gas was used for them, as their previous experiences of ether had been unfortunate. Despite all care preoperatively and at operation, these patients generally suffered from severe after-sickness, less only in degree from that

reported by them as following ether. No reasons could, in general, be assigned for this unpleasant response to anæsthetics. Clinically, the patients seemed to belong to either of two groups (*a*) a group giving a history of digestive instability, the patient being readily nauseated by certain foods or drugs, (*b*) a group in which the history pointed to possible labyrinthine disturbance, evidenced by car-sickness or train-sickness. Speculation, however, would prove less useful than biochemical investigation of these unfortunate individuals.

Reverting to the table, it will be seen that patients for abdominal operations showed much the same incidence of after-sickness as patients for general surgical operations under the same anæsthetic, a result which differed from that anticipated, as interference with the abdominal contents might be expected to produce much reflex vomiting. Patients habitually vomiting before operation were, of course, excluded from the series, as it was impossible to assess post-anæsthetic sickness in them.

Oto-rhino-larynological cases showed relatively little after-sickness, possibly because they involved no great laceration of tissue. Where ether was employed vomiting (mainly of blood) was usual, but was rarely severe. With gas anæsthesia recovery was rapid and swallowing of blood was minimised, so that the incidence of after-sickness was still less than after ether.

Minor dental cases were excluded from the table, their shortness and absence of tissue-trauma making them not comparable. Major oral surgical cases under endotracheal ether or gas anæsthesia were, however, listed as general surgical procedures. Actually, minor dental cases under ether were almost always followed by vomiting, mainly of blood. When gas anæsthesia was employed, vomiting was exceptional, perhaps because of the rapid awakening with little swallowing of blood. This may be illustrated by the following table.

Of 124 cases receiving short gas anæsthetics (by the nasal route) for dental operations —

Nitrous oxide-oxygen was used in 123 cases and ethylene oxygen in one

Ether supplement was required in 11 cases (9 per cent), but no vomiting ensued

Of 113 cases requiring no ether supplement, vomiting occurred in three only (2.6 per cent)

As remarked above, the series of cases was too small to render it profitable to analyse the separate operations. Evidence existed, however, to show that certain operations were associated with more consistent after-sickness than others. Ocular tendon-transplantation for strabismus in children showed a high incidence of vomiting, whether due to interference with the ocular musculature or to the mere fact of the children being obliged to awaken from the anæsthetic with their eyes bandaged. Operations for toxic goitre were likewise followed by more than the average incidence of vomiting, but this disturbance may be in part attributable to the routine, intensive, post-operative iodine therapy.

The fact that many patients are nauseated by certain sedative drugs is well known, and many such patients were encountered in the present series. Morphine and the more potent barbiturates, such as sodium amytal and nembutal, were the usual offenders, little or no disturbance was to be attributed to the less potent barbiturates such as barbitone. The writer's experience of paraldehyde as a basal narcotic, rectally administered, has been small, contrary to the usual impression, its use was followed by more than the usual amount of vomiting in those cases in the series in which it was employed.

The suggestion has been advanced that induction of "open" ether anæsthesia with the aid of carbon dioxide is associated with less after-sickness than is the case with the ethyl-chloride-ether "sequence". A small series of cases was available for the investigation of this claim.

Method of induction	Cases	No vomiting	Slight	Moderate	Severe
"Open" ether with CO ₂	112	23=20.5%	78=69.6%	8=7.0%	3=2.7%
Ethyl chloride- ether	517	121=23.4%	322=62.3%	64=12.4%	10=1.9%

It seems that, although the number of cases is small, little difference existed in the incidence of after-sickness, whichever method of induction was adopted

One case was encountered in which post-anæsthetic vomiting was the determining cause of death. The patient, an enfeebled man, aged 76 years, received nitrous oxide-oxygen-ether anæsthesia for single-stage removal of the prostate. Biliary vomiting began in the induction stage and persisted through the operation. Post-operatively, it reached such proportions that no fluid could be retained, and dehydration ensued. Rectal and intravenous administration of fluids were of no avail, the patient entered a state of histamine-like shock, to which he succumbed. The cause of the excessive vomiting is not clear, but it is possible that the operation happened to coincide with the onset of uræmia.

The writer has nothing to add to the accepted measures for averting or treating post-anæsthetic vomiting. He believes the taking of concentrated carbohydrate (cane-sugar, boiled sweets, barley-sugar) on the evening preceding operation to be helpful, although he has no statistics to prove this. Glucose has been found somewhat nauseating, and dextrose is preferable, especially for patients who do not like sweetstuffs. Once vomiting occurs, no remedy has been found generally helpful. The drinking of a drachm of sodium bicarbonate dissolved in a half-pint of water is sometimes useful, even if the mixture be returned by the patient. At other times better results have been given by the sucking of ice, the taking of iced dry ginger-ale or soda water, or of citrus-fruit drinks containing much sugar or dextrose. It is often best to allow the patient to make his own choice from the above measures. Post-anæsthetic vomiting, according to various nursing sisters who have been questioned, is certainly less nowadays than ten years ago, with which impression the writer would agree. The improvement is, perhaps, attributable to the modern trend in anæsthesia, which seeks muscular relaxation sufficient only for the needs of the moment, and spares the patient from prolonged, over-deep narcosis. Instead of the former pre-operative starvation, attention is now given to ensur-

ing an adequate reserve of liver glycogen, further, gas anæsthesia is employed wherever the hepatic functions are under suspicion. At the same time, post-anæsthetic vomiting is by no means controlled. Anæsthetists are apt to take it rather as a matter of course, but it is much dreaded by patients, and would justify the further laboratory and clinical studies required for its control.

Respiratory Sequelæ

The incidence of post-anæsthetic bronchitis and pneumonia in the above series of 2,520 cases is given in the following table. The latter does not include cases of transient exacerbation of frankly existing respiratory infection. No case of lung-abscess was encountered in the series.

Anæsthetic	Adminis- trations	Respiratory Sequelæ
Ethyl chloride	17	—
Ethyl chloride to "open" ether	360	1
"Open" ether	38	—
"Open" ether with CO ₂ induction	141	—
Endopharyngeal ether	394	1
Endotracheal ether	907	5
Chloroform	21	—
Chloroform to "open" ether	7	—
Nitrous oxide-oxygen	188	2
Nitrous oxide-oxygen-ether	124	3
Nasal nitrous oxide (dental)	124	—
Endotracheal nitrous oxide	85	1
Ethylene-oxygen	57	—
Ethylene-oxygen-ether	21	—
Endotracheal ethylene-oxygen	36	2
	2520	15*

* Equals 0.59 per cent

The actual operations followed by respiratory sequelæ are shown in the following table

Operation	Number performed			Respiratory sequelæ		
	Total	Ether, etc	Gases	Total	Ether, etc	Gases
<i>Abdominal</i>						
Gastric	23	9	14	1	—	1
Intestinal	96	66	30	—	—	—
Gall-bladder	63	29	34	1	—	1
Septic	4	—	4	—	—	—
Various	26	14	12	1	1	—
<i>Gynæcological</i>						
Pelvic	54	39	15	1	—	1
Vaginal	30	11	19	—	—	—
Emergency	2	—	2	—	—	—
<i>Genito-urinary</i>						
Bladder	15	4	11	—	—	—
Prostate	20	4	16	—	—	—
Renal	17	9	8	—	—	—
Various	18	12	6	—	—	—
<i>Thoracic</i>						
Empyæma	8	—	8	—	—	—
Thoracotomy	7	1	6	1	—	1
Others	3	1	2	—	—	—
<i>Thyroid</i>						
Toxic	44	—	44	—	—	—
Non-toxic	3	1	2	—	—	—
<i>Oto-rhinological</i>						
Tonsils and adenoids	679	634	45	2	—	2
Nasal sinuses	383	352	31	1	1	—
Various	246	212	34	4	3	1
<i>General Surgical</i>						
Bones and joints	85	55	30	1	1	—
Breast	22	8	14	—	—	—
Cerebral	39	13	26	—	—	—
Face and jaws	50	44	6	—	—	—
Herniotomy	82	73	9	1	1	—
Various	176	109	67	—	—	—
<i>Dental</i>						
	325	185	140	1	1	—
	2520	1885	635	15	8	7

In investigating the reasons for the 15 cases of bronchitis and pneumonia, climatic factors must be discounted, since three of the cases occurred in spring, seven in summer, five in autumn and none in the winter. The apparent reasons for the sequelæ may be classified as follows

(a) *Faulty Choice of Anæsthetic* Three cases

1 *Endopharyngeal ether* Bronchitic woman for laparotomy. Gas declined on economic grounds, chloroform unsuitable. "Warmed ether" was given. Exacerbation of bronchitis, recovery.

2 *Endotracheal ether* Bronchiectatic child for antrostomy. Chloroform unsuitable and gas not available in that hospital. Bronchopneumonia, recovery. Subsequently given endotracheal nitrous oxide for another operation without disturbance.

3 *Endotracheal ether* Woman with acute mastoiditis, high fever and rigors, believed to have lateral sinus thrombosis. Sinus found normal but virulent mastoid infection present. Next day had serous labyrinthitis with temperature 106°F. Overwhelmingly toxic pneumonia supervened, with death on third day. The condition may have been septicæmic from the start, the rigors heralding the onset of pneumonia. The administration of ether, however, must have determined a fatal issue.

(b) *Technical Errors* Four cases

1 *Endotracheal ethylene* Asthmatic male patient for tonsillectomy. Catheter unduly small and inadequately packed-off. Probable entry of blood into air passages. Mild bronchopneumonia, recovery.

2 *Endotracheal nitrous oxide* Old man for laryngofissure. Owing to an oversight, the pharynx was not thoroughly aspirated before withdrawing the endotracheal tube. The first deep breath after removal of the tube led to inhalation of blood into the air-passages. Death from bronchopneumonia 36 hours later.

3 *Endotracheal ether* Adult, male, confined to bed for three weeks by giddiness due to labyrinthitis complicating mastoid disease. Bronchopneumonia supervened on the third day after operation, with fatal results. The patient might have been saved had the complication been reported earlier and CO₂ therapy initiated.

4 *Open ether* Child, for open reduction of fractured forearm. Unduly long period of post-operative recumbency in splints. Mild bronchitis, recovery.

(c) *Various* Seven cases

1 *Nitrous oxide-ether* Old man for gastrectomy. Costal margins were infiltrated with novocain and were very sore post-operatively. Pneumonia, perhaps atelectatic, supervened, with recovery. Three weeks later, with the onset of colder weather, pneumonia returned,

the patient then suffered from three attacks of angina pectoris and succumbed to the last

2 *Nitrous oxide-ether* Young man with infected hydatid of lung and copious, purulent expectoration Anæsthesia difficult, because mask had to be removed periodically whilst pus was expectorated Pleurisy and pneumonia developed on the unaffected side on the sixth day after operation, due perhaps to the anæsthetic or perhaps to aspiration from the affected lung The patient recovered

3 *Endotracheal ethylene* A young man, suffering from rheumatic carditis, required tonsillectomy A mild post-operative pneumonia followed, for which the cause is obscure No obvious technical error existed to explain it

4 *Nitrous oxide-ether* An anæmic woman, suffering from arteriosclerosis and angina pectoris, required hysterectomy The operation was long and difficult Pneumonia occurred on the third day, with slow recovery The cause of it is obscure

5 *Endotracheal ether* A woman weighing twenty stone, and suffering from asthma and arteriosclerosis, required operation for incisional herniæ She had, in recent years, undergone 28 operations, nearly all followed by septic sequelæ On this occasion, the surgical wound sloughed and bronchopneumonia developed on the third day Recovery ensued The pneumonia was, in this case, probably atelectatic in origin

6 *Endotracheal ether* Healthy woman for dental extractions History of pneumonia after a previous anæsthetic On this occasion, developed mild bronchitis, from which she made an uneventful recovery The ætiology is obscure

7 *Nitrous oxide-ether* A child, gravely ill, suffered from double acute mastoiditis Pneumonia developed after operation, but, as this was of lobar type, it was probably related to the disease-condition rather than to the anæsthetic

The fifteenth case of respiratory infection merits separate description, as it was the one case in the series in which the anæsthetic was the directly determining cause of death

A woman, aged 78 years, suffered from abdominal pain thought to be due to cholecystitis On the morning of operation she vomited profusely, but this fact was not reported by the nursing staff Anæsthesia was induced with nitrous oxide-oxygen with a small supplement of ether When the surgeon palpated the abdomen prior to incision, a tide of bile-stained fluid welled up and flooded the patient, respiration ceasing at once Resuscitation was affected, but the operation was abandoned, it being thought that the patient's only hope of avoiding pneumonia now lay in having an intact abdominal wall, permitting of coughing Carbon dioxide therapy was at once initiated The patient, whose expectoration was bile-stained, soon showed respiratory

embarrassment, and died three hours later from acute congestion of the lungs. The revised diagnosis of cholecystic malignancy and gastric dilatation was confirmed at autopsy, revealing a malignant growth of the gall bladder adhering to the transverse colon and pressing on the duodenum. The lungs were congested and hæmorrhagic.

This fatality, although scarcely to be regretted in many ways, would have been directly preventable had the gastric dilatation been recognised and endotracheal anæsthesia adopted.

Of other possible respiratory sequelæ of anæsthesia, three deserve mention, viz pulmonary embolism, aggravation of existing phthisis and laryngeal trauma. Of the first, no statistics have been collected by the writer, as the evidence does not point towards any relation between this complication and anæsthetic technique employed.

Where pulmonary tuberculosis is diagnosed, gas anæsthesia is commonly employed. Unfortunately, the condition may not be recognized and ether be given, with harmful results. Two such cases were noted in the above series. In the first, an apparently healthy man presented himself for operation for acute mastoiditis. A month later his condition necessitated bilateral radical mastoidectomy. Four months later it was necessary again to operate, when the condition found indicated tuberculous mastoiditis. Death occurred several months later from tuberculous meningitis. The second patient, also apparently healthy, underwent a dental operation, likewise under endotracheal ether anæsthesia. Post-operative pyrexia led to further investigation, which revealed on X-ray gross bilateral pulmonary consolidation, for which almost no physical signs were apparent. The patient was transferred to a sanatorium. In these two cases, then, a latent tuberculous infection was overlooked, and ether was administered to the patient's detriment. How often the same thing may have happened in other cases in the series it is, of course, impossible to determine.

The incidence of laryngeal trauma after endotracheal anæsthesia has been less carefully worked out in this series than should have been the case. Some measure of sore throat, lasting a few hours only, seems to be a not uncommon sequel, although the patient may not refer to it unless specifically questioned. More serious laryngitis or

tracheitis was occasionally observed. Certain cases could be accounted for by trauma to the pharynx or epiglottis, the parts being contused by the laryngoscope either from haste, carelessness or the difficulty of exposing an unfavourably-placed aditus. Although usually transitory, the resulting sore throat lasted in one or two instances for several days. No case of permanent laryngeal damage has come under the writer's observation. In certain cases, however, laryngitis followed perfectly easy intubations, and no obvious cause for it could be found.

In an attempt to ascertain the frequency of laryngeal sequelæ in the above series, a group of 518 records of endotracheal administrations was analysed. No special inquiry was made as to sore throat or hoarseness, the presence of these would only be mentioned on the record if their severity were such as to lead the patient to complain of them. The results may be set out as follows.

Complication	Endotracheal ether by inhalation using wide-bore rubber tubes 269 cases	Endotracheal ether by insufflation through small tubes 192 cases	Endotracheal nitrous-oxide or ethylene, by inhalation through wide- bore tubes 57 cases
Hoarseness without pain	1	1	—
Definite laryngitis or tracheitis	3	1	2
Pharyngeal ulceration	4	—	—

Of the laryngitides, one was clearly due to trauma to the epiglottic region in a case in which exposure of the larynx was practically impossible. The two laryngitides after gas anæsthesia both occurred in cerebral cases, intubation was perfectly easy in one, but the presence of a large tube in the larynx for several hours may have determined the

laryngitis * The other case was probably due to some measure of trauma. Incidentally, of the 57 endotracheal gas administrations, 22 were for cerebral operations. The other three cases of laryngitis were obscure, as no undue difficulty was met in intubation. The cases of pharyngeal ulceration were due to trauma from the mouth-pack or post-nasal plug, either as a result of over-enthusiastic packing by the surgeon or from the use of dry packing gauze, instead of gauze moistened or lubricated with paraffin.

In summary, the incidence of major respiratory sequelæ of anæsthesia in hospital practice seems to have lessened materially since about the year 1928. The writer cannot advance statistics to prove this, but his impression is shared by ward-sisters, who are generally in a position to know. Previous to 1928 respiratory infection was quite common, and the writer is confident that his personal record before that year, had adequate records been kept, would have proved a bad one. The recent improvement has, in all probability, several causes. Better care is taken to safeguard patients from exposure and freer use is made of gaseous anæsthetics in cases where they seem to be desirable. Endotracheal anæsthesia in appropriate cases has become a commonplace of hospital practice, almost every resident medical officer gaining experience in its administration. Finally, unnecessarily prolonged and deep anæsthesia is in disfavour with both surgeons and anæsthetists, whilst full use is made of post-operative inhalations of carbon dioxide.

Other Undesirable Sequelæ

(1) *Post-operative psychosis* The number of cases in the present series was not recorded, but was probably five or six. The onset occurred usually several days after operation, and bore no apparent relationship to the anæsthetic technique. The writer has an impression that permanent psychotic changes are rather frequently encountered after the operation of prostatectomy.

* As a result of a recent unfortunate experience in the same operating theatre, it is quite probable that laryngitis was due in this case to the tube not having been cleansed of carbolic before being handed to the anæsthetist.

One case of post-operative psychosis was fatal, although it actually occurred in 1928, before the period of the present report. A middle-aged woman underwent an operation for vaginal repair and sterilization under "open" ether anaesthesia. The course of the operation was uneventful. Next day an acute sexual psychoneurosis developed, leading to death from exhaustion. Autopsy revealed no pathological lesion. Inquiry into the private history disclosed a tragic disharmony of sexual life, leading to a pre-psychotic state for which the gynaecological operation acted as a trigger-release.

(2) *Sore eye* One patient in the series suffered from corneal abrasion. He had struggled during the induction stage and either received a splash of ether in the eye or, more probably, was injured by the gauze face-cover. In times past the writer saw several cases of "ether sore eye". Most of them were due, not to entry of ether, but to neglect to see that the lids remained closed during anaesthesia. The exposed cornea was then abraded by the face-cover.

Some Notes on Moot's Index

Moot's index³ is one of the best-known tests for anaesthetic tolerance. For some years it has been the standard test at the writer's hospital in guiding junior resident medical officers in assessing circulatory reserve. Recently it has received some criticism from cardiologists, who have stated that the index does not adequately represent the reserve of cardiac patients as shown by clinical and electrocardiographic findings.

From the anaesthetist's standpoint the test would have value if it would help in forecasting the degree of circulatory depression likely to occur in a given patient during a particular operation and type of anaesthesia. To work this out would have required a larger series of cases than the writer possessed. Unfortunately, the Moot's index is usually calculated when the patient is seen on the evening before operation and has been frequently omitted from the record when the latter is made up on the following day. In consequence, only 693 complete records were available. It was decided to group these according to the Moot's index and to the

anæsthetic risk, in the hope of being able to say whether, given a certain degree of anæsthetic risk, the presence of an abnormal Moot's index would imply a greater liability to circulatory depression at operation

The results are set out in the following table *

Moots' Index	"A"-Risk			"B"-Risk			"C"-Risk			"D"-Risk		
	Degree shock*			Degree shock			Degree shock			Degree shock		
	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd
25 or under	79	3	—	19	11	—	15	14	2	—	5	2
75 or over												
26-39, 61-74	131	3	—	34	8	1	7	10	—	3	3	—
40-60	215	14	1	57	18	1	22	10	1	2	—	2

* Degree of shock was assessed by McKesson's classification, and represents the greatest circulatory depression observed at any stage of the operation, irrespective of the patient's condition at the end of it.

This table, as it stands, is not particularly informative. The data may be more usefully arranged in terms of the anæsthetic risk, as in the table which follows

446 "A"-Risk Patients

Moot's index	Incidence per cent	Degree of shock seen in those cases		
		1st degree	2nd degree	3rd degree
25 or under	82 cases or 18.4%	79=17.7%	3=0.7%	—
75 or over				
26-39, 61-74	134 „ 30.0%	131=29.4%	3=0.7%	—
40-60	230 „ 51.5%	215=48.2%	14=3.1%	1=0.2%

We infer, then, that the majority of "A"-risk patients had indices between 40 and 60, as might be expected. The incidence of shock in such patients was small, as one would expect from the healthy condition of the patients and the minor nature of the operations. It is interesting to note, however, that abnormal Moot's indices, below 25 or over 75, were relatively frequent in "A"-risk patients, and did not

seem to imply any special liability to circulatory depression at operation

It will now be helpful to apply similar analysis to the "B"-risk patients

149 "B"-Risk Patients

Moot's index	Incidence per cent	Degree of shock seen in those cases		
		1st degree	2nd degree	3rd degree
25 or under } 75 or over }	30 cases or 20.1%	19=12.8%	11=7.4%	—
26-39, 61-74	43 .. 29.0%	34=22.8%	8=5.4%	1=0.7%
40-60	76 .. 51.0%	57=38.2%	18=12.1%	1=0.7%

Here we find that the incidence of circulatory depression much resembles that encountered in "A"-risk patients, although the occurrence of shock of the second and third degree has relatively increased. The presence of an abnormal Moot's index was frequent, although this did not imply any particular liability to circulatory depression.

We may now apply a similar classification to the "C"-risk cases.

81 "C"-Risk Patients

Moot's index	Incidence per cent	Degree of shock seen in those cases		
		1st degree	2nd degree	3rd degree
25 or under } 75 or over }	31 cases or 38.3%	15=18.5%	14=17.3%	2=2.5%
26-39, 61-74	17 .. 21.0%	7=8.6%	10=12.3%	—
40-60	33 .. 40.7%	22=27.1%	10=12.3%	1=1.3%

We see here that the Moot's index still lay most usually between 40 and 60, but that the incidence of abnormal indices had increased relatively to "A"- and "B"-risk patients. Further, an increasing percentage of patients showing an abnormal index suffered from circulatory depression of the second or third degree at operation.

The number of "D"-risk patients was so small as to make a table of questionable value. It is given, however, for what it is worth.

17 D-Risk Patients		Degree of Shock seen in those Cases		
Moot's Index	Incidence per cent	1st Degree	2nd Degree	3rd Degree
25 or under	} 7 cases or 41.2	—	5=29.4	2=11.8
75 or over				
26-39, 61-74	6 „ 35.3	3=17.6	3=17.6	—
40-60	4 „ 23.5	2=11.8	—	2=11.8

In this very small series an abnormal Moot's index was usual, and was in each case followed by more or less severe circulatory depression.

If one may draw conclusions from a series of 693 cases, one would say that an abnormal Moot's index was quite a common finding in patients who were clinically excellent "anæsthetic risks." In them it did not seem to have any serious significance. One is tempted to conclude that Moot's index may be ignored when it is found to be abnormal in clinically healthy patients, but that an abnormal index in a patient who is a "bad risk" on clinical grounds should be regarded seriously, because it seems to imply circulatory inefficiency. In other words, Moot's test can supplement, but not replace, a clinical assessment of the patient's condition. It would be interesting to submit others of the well-known tests for circulatory reserve, such as the breath-holding test or the energy index, to similar investigation.

Conclusion

The writer has presented an analysis of 2,520 administrations of inhalational anæsthetics, recognizing that his experience in other forms of anæsthesia does not entitle him to hold any positive views in regard to them. The cases that have been presented have been followed up and worked out as fully as possible under the conditions in which the writer works. The series is too small for any dogmatic statements to be based upon it, and the writer has tried not to strain conclusions further than their evidence will

admit If the results have been in some respects satisfactory, they have in other directions given no cause for pride They have, however, been reported without reserve, because the frank acceptance of bad results must be the first step in their future prevention It is the writer's hope that other anæsthetists, of wider experience and with greater facilities, will similarly report their statistics In this way it should be possible to form a true picture of the incidence of undesirable sequelæ of anæsthesia, and of the most effective means of combating them

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MODERN TREATMENT BY HYPNOTICS AND BASAL NARCOSIS

From the French of H. WEESE, *Anesth. et Analges.*,
February 1936

THE simple effect of narcotics and of hypnotics is to modify physiological reactions or reactive equilibria. If we are to demonstrate their behaviour as a group and to appreciate their value, we must know the physiology and the pathology of sleep itself. Sleep is a physiological phenomenon in which the routes of afferent and efferent excitability are interrupted and in which the diminished excitability of the autonomic nervous system leads to profound extinction of consciousness, that is, the cessation of relations with the exterior world. Fatigue is the preliminary condition most often necessary for the advent of sleep. The seat of this fatigue is the central nervous system, and consequently the phenomena of sleep are the more defined the more developed is the central nervous system. Man passes one-third of his life in sleep. The normal daily sleep of an adult is from six to eight hours. Its depth varies in a manner which is completely characteristic. It is determined by the intensity of stimulant necessary for awakening. Healthy sleep reaches its maximum depth quickly and becomes lighter after an hour or two. The subjects of nervous disorder never attain really deep sleep.

The joint depth and duration of sleep are designated by Frey its "quantity," and this constitutes a measure of the restoration brought about. After great physical effort as well as after a suitable hypnotic the "quantity" can be doubled or trebled by increased profundity of the sleep without its duration being prolonged. As the excitability of the respiratory centre is diminished during sleep in proportion to its depth the tension of CO_2 in the blood, and consequently in the alveolar air, is increased. Determination of

the depth of sleep by estimating the carbonic acid of the alveolar air gives results analogous to those obtained by ending the sleep by means of a measured stimulant. Physiologically sleep appears under two conditions, fatigue and suppression of stimuli. The former hypotheses, according to which brain as well as muscles became fatigued under the incomplete metabolism, particularly of lactic acid or of carbonic acid, endowed with hypnotic action, as Du Bois maintained from observation of hibernating animals, can no longer be admitted. We have rather to think of products, still unknown, of the disintegration of proteids. The moderate acidosis which exists during sleep is not a cause but a consequence of the lowered excitability. Sleep, however, can come on without corresponding labour and consequently without fatigue. The latter is one of numerous preliminary conditions.

Actually sleep is produced by the fact that there is an internal necessity in the mass of cells whereby phases of rest must be introduced into the activity of the organism. Thus one can affirm that sleep is not a purely passive phenomenon, a reaction with regard to some substance or other, but, on the contrary, it is the consequence of a counter-regulation, of a process of excitation, in short, the expression of an activity. The symptoms of sleep are a clear proof of this. In animals one can recognize, in fact, on the part of the sleeping subject muscular contractions such as the prehensile reflex of birds and the upright posture of the horse. Man presents also muscular contraction of this nature, such as contraction of the pupils, upward deviation of the eyes, and the closure of sphincters. W. R. Hess in recent years has made a searching analysis of the active and passive processes which supervene in the sleeping subject. During sleep motoricity is checked in its relationship with conscious processes, for a series of deep reflexes is extinguished. The arrest of motoricity must consequently be looked on as an autonomous symptom of sleep. Respiration and heart-beat are slowed. The excitability of the respiratory centre is lessened. The potassium and calcium of the blood are lowered. The production of urine is diminished. Digestive functions persist in so far as they

are not dependent on conditioned reflexes, that is to say, allied to consciousness

We find, consequently, in sleep an important selective diminution of animal activities. Among the vegetative functions few are reduced. The reason is, according to Hess, that the relative incapacity which prevents the sleeping subject from perceiving and appreciating external stimuli can be overcome after a few seconds, or at most a few minutes. This inhibitory power of sleep shows, therefore, a regulated selection with regard to certain organs or functions, and represents, in short, an adaptation to the momentary conditions of sleep. This tends—and here, according to Hess is the essential point—to a positive activity in the interest of the individual, having for its object to favour the processes of cellular restoration. After a refreshing sleep the individual possesses a new potential and new reserves in the widest sense of the word. Nevertheless, one is active during sleep.

The pharmacologist is tempted to consider narcosis as a state of profound sleep during which the processes of repair ought to be strengthened, or at least persist without modification. The contrary is observed: during narcosis these processes are diminished. From this point of view narcosis constitutes an inversion. Sleep and narcosis are, it is true, essentially identical, but the curve of restitution oversteps its optimum when deep sleep passes into narcosis.

The teleological order which exists in sleep shows that some apparatus functions in the course of this phenomenon. As opposed to the sympathetic, the parasympathetic regulates chiefly the economic restorative and protective functions of the organism. W. R. Hess considers that it is this which is the motive power of sleep. The regulation of this can be conceived only in the shape of an organic structure. The approximate localization of this controlling centre has been the natural object of a mass experiment carried out by *encephalitis lethargica*. It is characteristic of this complaint that, alongside of troubles affecting the visual apparatus, there are observed very severe ones which affect sleep, for example persistent somnolence, sleep inversion, dissociation between cerebral and bodily sleep, and, finally, sleepless-

ness Economo has consistently found, in the brain of subjects dead of encephalitis, inflammatory alteration of the brain matter at the junction of the diencephalon and the mesencephalon. Where there was persistent sleep the lesion chiefly concerned the caudal portion, and when there was insomnia the anterior portion of this region. Economo has therefore conceived the notion of a sleep regulating centre seated in the cerebral peduncles. Hess has established by experiment the existence of this sleep-regulating centre.

For this purpose he introduced fine electrodes into the brains of cats. Electric stimulation of certain regions enabled him to call out all the symptoms of going to sleep: the animals yawned, took up the posture of rest, and then fell asleep. Hess has then succeeded in lifting the latch of sleep by an artificial stimulus. The cerebral regions, the stimulation of which allowed of this physiological experience, are situated in the grey nuclei of the base, but in front of those the inflammation of which causes pathological somnolence. One is forced to admit that this regulating centre excites the vagus which is awake during sleep. Hess has been able to bring about the predominance of vagal tone by weakening the tone of its antagonist, the sympathetic. Actually it has been possible to put animals to sleep by injecting into the third ventricle ergotamine, a substance devoid of hypnotic action. Economo and Hess consider that it is from this region of the grey matter that sleep is primarily and directly ordered. These authors do not, nevertheless, believe that this collection of ganglionic cells is alone capable of causing sleep. There are reasons to believe that the regulation of sleep is, in fact, ubiquitous and diffuse. It is by the mediation of the vagus that the regulating centre brings on bodily sleep. An inhibition of the hemispheres, starting from this centre, almost completely suppresses consciousness, and an inhibition of the diencephalon interrupts the routes of stimulation and induces cerebral sleep. Our desire for a causation is, nevertheless, not satisfied by the results of the most recent research. It is now a question of knowing the cause of the stimulus. We have already alluded to the "internal rhythm" independently of which one has to admit

a chemical stimulus of the regulation of sleep, a stimulus which will be the consequence of fatigue

Piéron in 1913 demonstrated the existence of "ponogenous" substances. Legendre and Piéron were able to prevent a dog from sleeping for several days, and on injecting the blood-serum, cerebral extracts, or the cerebro-spinal fluid of this animal into the fourth ventricle of normal dogs at rest they were able to bring on somnolence and sleep. Their hypnotoxins apparently possessed the following properties: they are soluble in water, insoluble in alcohol, not dialysible or ultra-filtrable, destroyed by oxygen and by heating to 65°C . Marmesco as well as Demole has been able to induce artificial sleep by injecting small quantities of cholin or of CaCl_2 in the neighbourhood of the tuber cinereum, and Cloetta has proved that during sleep there is in this region a slight increase in the calcium content. This enrichment in a specific region shows that it is not the question of a cause but simply of a concomitant local symptom or of a consequence of sleep without which the pathological or therapeutic modifications of the blood-calcium would be accompanied by corresponding symptoms of sleep. Jondeck and Bui have proved that during normal sleep or that due to drugs or illness there is an increase of bromine in the region of the centres of the diencephalon and at the same time diminution of it in the hypophysis. They concluded that in the course of this condition a bromo-hormone passes from the hypophysis to the diencephalon. Pincussen, however, showed that their evidence of bromine was not accurate, and the estimation of this hormone of sleep remains at the least doubtful.

Although we do not yet know the physiological ponogenous substances, we must, nevertheless, admit that the regulation of sleep acts on them and protects the other organs against an invasion by these substances. According to Claperède "we sleep not because we are intoxicated by ponogenous substances but in order to protect ourselves against them." Physiology has not hitherto provided any foundation permitting us to realise in cases of insomnia a substitution therapy. Most fortunately, however, we have succeeded in purely empirical fashion in practising a phar-

macological therapy of sleep We must, it is true, renounce for the time being the induction of sleep by administration of physiological substances, but we are able to satisfy the second of the conditions necessary for sleep, often the most difficult to realise, by the suppression of stimulation This success is achieved by attenuating temporarily the senses and central excitability by means of hypnotics and narcotics There is no question here except of the so-called "indifferent" narcotics, narcotics which, as opposed to certain alkaloids acting in an analogous manner, do not enter into chemical reaction with the cells responsible for hypnosis and narcosis Many hypotheses have been put forward in the hope of enabling us to capture the mechanisms by which hypnotics and "indifferent" narcotics work, and to enable us to find new substances of this kind

Among all the theories of narcosis the lipid theory of Meyer and Overton has remained the most fruitful to the present time This theory affirms that only substances chemically indifferent, soluble in lipoids, can have narcotic action on the protoplasm Solution of the narcotic in the cellular lipoids is the principal condition of their specific activity which is shown the more quickly and energetically the richer the cell is in lipoids The relationship between their lipid and their aqueous solubility determines the activity of narcotics Meyer and Overton measured this relationship in an olive oil-water system Since the triglycerides possess no physiological significance in the nerve cell there has always been justifiable mistrust of the experimental basis of the lipid theory This mistrust was strengthened when Traube had shown that it was the surface activity, the adhesion pressure (surface tension) which corresponded with narcotic effect, not the relative solubility. Traube's capillary tube experiments do not reproduce living conditions sufficiently exactly to carry conviction Traube's theory demands, in opposition to that of Meyer and Overton, a certain degree of independence between the structure of the cell and its sensibility to narcosis Traube's theory has been developed by Warburg This author insists still more on the part played by the structure of the cell He has been able to show the inhibition by narcotics of oxidation at the

surface of cystine, for example. In consequence, he contends that the surface at the level of which enzymes exercise their catalytic activity should be invaded by narcotics. So far as inhibition of oxidation is concerned, he has found a formula which permits, given the constants of absorption and of molecular volume of a narcotic, the foretelling of its activity in oxygen inhibition. Warburg has further shown that with narcotics one can succeed in inhibiting fermentation of yeast.

Warburg's theory has been subjected to criticism by Meyer and Hemnis. They rightly insist on the fact that in the living cell there is no surface so clearly defined as that of charcoal used in experiments. In this the atoms are joined together in a manner to offer resistance to the penetration of the substance much more effectively than can the surfaces, steeped in lipoids, of the proteins and carbohydrates. These authors also demonstrate that the concentration of narcotics capable of inhibiting yeast fermentation coincides very exactly with that which precipitates nucleoproteids. But as this concentration is of an order very much greater than that which can cause narcosis in tadpoles, and as further oxidation of cystine in the presence of carbon is of an order much less than that of the tadpole experiments, Meyer contests that the same phenomenon is concerned in every instance.

Warburg's experiences have shown, it is true, that at the level of surfaces oxidations are inhibited by narcotics according to a definite law. But no experiment yet performed on an animal can show that narcosis results from inhibition of oxidation in Warburg's sense. Still more Schlossmann has shown that tissue exchanges are scarcely inhibited by narcosis. Anoxæmia can certainly induce a condition like enough to narcosis, but the phenomena are quite different. Winterstein, truly, finishes his book on narcosis with the conclusion "the mechanism of action of narcotics is founded on the fact that these substances are easily absorbed by the structural elements of the living organism." Nevertheless, the hopes which were founded on the theory of absorption have not been realised. Meyer and Henuni have striven anew to consolidate the foundations of the lipoid theory. In their

researches they have directed themselves to the weak point of the lipid theory, that is to say, to the discovery of a substance which can truly provide a representative of the organic lipoids. As the cerebral lipoids contain, alongside cholesterol which possesses but one hydroxyl group, substances such as cerebrin or kersin which possess several, they have chosen an indifferent substance, olein-alcohol, in which the HO and HC groups present intermediary relations. By tadpole experiments they succeeded in deducing from the relationship between olein alcohol-water and the narcotic concentration confirmation of the Meyer-Overton theory which, according to them, would be still more exact if one could find a still better representation of the organic lipoids. Traube, in his answer, draws attention to the fact, which he demonstrated by the behaviour of various alcohols, that the lipid solubility besides other physico-chemical properties of narcotics must play a part sometimes decisive.

A satisfactory theory of narcosis cannot be formulated without taking into account the sum of the physical and chemical properties of narcotics. Not one of the current theories deserves to be called a theory of narcosis, for not one of them permits deep penetration into the nature of the phenomena or represents more than a simple rule, a manner of reducing to a formula a collection of facts. The nature of narcosis has been brought nearer to us by the researches of Vernst, Hober and others on the modifications of permeability. We can put trust in the work of Winterstein. This author has succeeded, using the membrane of muscle, in showing that under the influence of an indifferent narcotic there supervenes a reversible modification in the permeability of the membranes for water and for substances soluble in the water. This phase corresponds to the reversible diminution of excitability which appears during narcosis. Stronger and more poisonous concentrations bring on an irreversible increase of permeability and in consequence definite tissue lesions. The work of Schlominsky and of Adler, who have been able by weak galvanic currents either to institute a true electric narcosis or to deepen narcosis brought about by indifferent narcotics, speaks in the same sense. Look at theories of narcosis how we will, the fact

remains that the concentration of the narcotic in the central nervous system is ruled by that of the blood. The concentration in the blood is the result of rapidity of absorption and of its contrary, detoxication. There results the falling asleep, the sleep of long duration or the narcotic effect according to the relationship between absorption and elimination.

[*To be continued*]

THE DIPLOMA IN ANÆSTHETICS

THE following are the names of the candidates who were recommended by the Examiners for the Diploma after the May examination

Beaver, Robert Atwood, B M , B Ch (Oxon), L R C P , M R C S
 Blatchley, Donald, M B , Ch B (Edin)
 Canwarden, Henry, L R C P , M R C S
 Carman, John Ambrose, M D , B S (Lond), L R C P , M R C S
 Coden, Bernard, M B , Ch B (Edin)
 Daplyn, Phyllis Frances Lucy, L R C P , M R C S
 Dawkins, Charles John Massey, M B , B Ch (Camb), L R C P ,
 M R C S
 Deane, Maslen Mackenzie, M B , B S (Melb), M R C S
 Galbraith, Margaret Louise Agnes, M B , Ch B (Birm), L R C P ,
 M R C S
 Grant-Whyte, Harry, M B , Ch B (Cape Town)
 Goldman, Victor Abraham, L R C P , M R C S
 Havers, Geoffrey Gordon, L R C P , M R C S
 Hudson, Maurice William Petre, M B , B S (Lond), L R C P ,
 M R C S
 James, Ian Clinton, M B , B S (Melb)
 Jones, Ivor Davenport, L R C P , M R C S
 Jones, Roland Neville, M B , Ch B (Birm), L R C P , M R C S
 Kennedy, Agnes Winifred O'Dwyer, L R C P , M R C S
 Mallinson, Frank Barnett, L R C P , M R C S
 Mansfield, Ruth Evelyn, L R C P , M R C S
 Nagle, Patrick Joseph, M B , B Ch , N U I
 Overton, Percy Michael, M B , Ch B (Leeds)
 Picken, Christopher Barugh, M B , B S (Lond), L R C P ,
 M R C S
 Rice, Raymond Arthur Cracroft, M B , B S (Lond), L R C P ,
 M R C S
 Woolf Abraham David, L R C P & S (Edin), L R F P & S
 (Glas)
 Wynter, Tamsin Mary, M D , B S (Lond), L R C P , M R C S

We much regret that the names of Messrs R E Apperly and H P Crampton were accidentally omitted from our former lists of those who had been granted the D A without examination

ABSTRACTS.

"*Anæsthetics for total pneumonectomy*" (W F REINHOF, in *Archives of Surgery*, February, 1936, p 230

After describing the surgical technique of the operation, the author discusses the question of anæsthetic. He used avertin supplemented by nitrous oxide and oxygen. The anæsthesia is kept very light. Whether or not an intra-tracheal tube should be used is, he thinks, an open question. "It cannot be denied that the insertion of a tube through the mouth into the trachea must always carry organisms into an otherwise sterile region." Also he maintains that damage to the lining of the trachea would produce a mucopurulent exudation and interfere with the movements of cilia. These factors tend to break down the protective mechanism of the patient. He gives details of a case in which he believes that unskilled passage of the tracheal tube set up damage which, resulting in lobar pneumonia, led to the death of the patient. The simpler the administration of the anæsthetic, the better, is his opinion.

"*Post-anæsthetic leucocytosis*" (E M BOYD, in *Canadian Med Assoc Journ*, February, 1936, p 159

Anæsthesia produces in man a leucocytosis independent of the surgical procedure. The leucocytosis reaches a maximum within three to four hours, and is probably due to migration into the blood of cells from liver, spleen, bone marrow, and other tissues. Microscopically, the leucocytosis is indistinguishable from that due to fever or infection, but the chemical composition of the white cells is entirely different from that in fever, late post-operative states, and infection. The effects of anæsthesia on the lipid composition of the white blood-cells was determined in 19 cases. It was shown that the polymorphonuclear leucocytes mobilized into the circulating blood after anæsthesia contain decreased amounts of phospholipid, about the same percentage of free

cholesterol, and a medium value for cholesterol esters and neutral fat, as compared with the leucocytes present before anæsthesia

"*Polyneuritis following evipan anæsthesia.*" (G B PALMER, in *New Zealand Med. Journ*, February, 1936, p 21

A woman, 28 years of age, the subject of recurrent melancholia but with no organic nerve defect, was given 0.9 grm evipan in order that a diagnostic curettage might be performed, as she suffered from menorrhagia. The operation was completed without any unusual symptom. Three days later she complained of poor sight. There was paresis of the right external rectus of the eye, weakness of the left side of the face, especially at the angle of the mouth, and partial paresis of the left side of the palate. After another three days the left external rectus was also paretic. Three months later the paralysis had almost entirely disappeared.

"*General anæsthesia with a new ethyl chloride, 'Novanest'*" B E ERIGO, P LINGI and R GILDO in *Ecce Ital di Anest e. Analges* December, 1935, p 505

The report is based on the use of novanest for 50 operations which included laparotomies, nephrectomies and operations on central nervous systems. The authors find no contra-indication, except broncho-pulmonary lesions or myocardial defect, to the use of this new inhalation anæsthetic which they say acts quickly, producing little excitement, causing no after-effects, and having no deleterious effect on any of the main systems of the body. The agent is given gradually on an Esmarch mask, about 10 c.c. being needed for induction of anæsthesia, after which minute quantities are added at frequent intervals. Recovery follows quickly after the administration stops. The blood-pressure, which rises at first, falls later, but only within narrow limits, and never gave rise to anxiety. The new drug has been prepared by Dr L Tirelli, who has conducted extensive chemico-physical researches upon it. The authors do not state its exact composition, but mention ether and chlorinated methylene in its production.

"Physiological and therapeutic action of carbon dioxide inhalation" H RIVERS and R. B GONZALES in *Gaceta Med le Caracas*, November 30th, 1935, p. 339.

The authors regard the introduction of carbon dioxide inhalation into the practice of surgery as one of the most valuable innovations of the day. They give a long account of the action of the gas as illustrated by experiments on animals and in its effects on the human subject, and lay due stress on its importance as a respiratory regulator. The production of acapnia by deficiency of CO_2 is described. They attribute the majority of accidents during inhalation anæsthesia to perturbation of the respiratory centre which is due, in their opinion, either to acapnia or to toxic elements. In this connexion they discuss atelectasis, reflex broncho-constriction, and reflex vasomotor dilatation of the bronchial vascular system, none of which factors can, they believe, be regarded as causing the deaths under discussion. They regard hyperventilation by CO_2 inhalation after operation as the chief means of avoiding post-operative lung complications.

"Making ether an ideal anæsthetic" W W KEMP in *Canadian Med Assoc Journ*, April, 1936, p 409

It is generally admitted that in the great majority of operations ether suitably given can provide perfect conditions for the surgeon. It is the patient's troubles afterwards that make the anæsthetist chary of employing ether as a routine. These troubles, Kemp believes, can be entirely or largely eliminated by proper pre-operative care and treatment. Based on experimental and clinical grounds his recommendations are (1) At least three days' stay in hospital before operation, (2) ten minims of Lugol's solution with six grains of desiccated suprarenal cortex daily for five days before operation, (3) for a week before operation a diet which is without meat and has high carbohydrate content and abundance of calcium and vitamins, and 15 ounces of lactose daily. Routine removal of mucus at close of operation, by endotracheal suction, is also regarded as one of the important measures in controlling post-operative nausea and sickness.

"Lesions of the nervous system from barbiturism"

J Shermitee in *Anæsthésie e Analg*, February, 1936,
p 1

Increasing use of the barbiturates by anæsthetists and of suicides from veronal, luminal, etc., have, says the author, drawn attention to the modifications of the nervous system provoked by barbituric intoxication. After massive introduction of barbiturates, when the acute symptoms have passed, there may be left mental troubles accompanied by ocular paralysis pointing to peduncular origin, or a clinical complex resembling that of cerebral tumours or even of disseminated sclerosis. Unfortunately, post-mortem and histological investigations have not equalled the clinical observations, for the former have commonly not fallen into the hands of those qualified to carry them out expertly. Authorities differ as to the region of the encephalon on which falls the brunt of barbituric intoxication. The author concludes that in massive intoxication it is impossible to localize the lesion, the whole central nervous system being subjected to a "brutal toxic submersion," but in subacute intoxications it may be possible that the lesions may carry the mark of a specialized pathological assault, and that this is in the regions most rich in myelinated fibres.

"Epidural anæsthesia." C ODOM in *New Orleans Med Surg Journ*, April, 1936, p 624

The author reports highly favourable results in 100 subjects. He reviews the origin of epidural anæsthesia, the anatomical facts involved and the work of Dogliotti. He describes an arrangement whereby it is made easier for the anæsthetist to be certain when his needle has entered the epidural space and that he has not penetrated the dura and gone into the cerebro-spinal fluid. This doubt really constitutes the main difficulty in using epidural analgesia, a method which has obviously great advantages of safety and wide applicability. It can be used for high regions of the body without the risk necessarily run when endothelial analgesia is employed for the same purpose. The anæsthetic employed is two per cent novocain solution and generally 50 c c represent the highest amount required.

"Importance of oxygen and of precise dosage of CO₂ in anæsthesia" R. MONOD and H. ARUAL in *Anesthe et Analges*, April 1936, p. 244

In a long article advocating certain alterations in the Ombrédanne inhaler and other closed apparatus, the authors discuss the physiology of carbon dioxide in anæsthesia. They quote authorities to show the baneful effects on the liver cells of too high a concentration of the gas, and believe that post-operative vomiting and post-operative mortality in diabetics are attributable to the same cause. In advocating the use of continuous or frequent small amounts of oxygen during anæsthesia, they believe that impoverishment in oxygen more than counterbalances the advantages of semi-closed methods. With regard to the use of CO₂, they state that though it may often be used to hasten the induction it should rarely be introduced in the course of anæsthesia. They are in accord with British authorities in seeking to avoid cyanosis entirely throughout operation.

REVIEW.

"Practical points in anæsthesia" H K ASHWORTH, M B
pp 160; illustrated Price 7s 6d. (J & R Churchill,
London)

This is a small volume likely to be of great use to the student who is undergoing practical instruction in the administration of anæsthetics. Although the more complicated methods and apparatus are not dealt with, yet the instructions given regarding those procedures which are most likely to be employed in actual practice by the average practitioner are clearly stated, and with that attention to detail which is so important if practical advice is to be of real use. The Sankey method of induction, which is approved of by the author for routine use, will be new to many readers, it differs in detail from the CE, open E, method which was for years so widely used in several London schools. The author gives a chapter to basal narcosis and one, supplied by W H T Simmons, deals with local methods of analgesia.

